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Medical and Surgical Reports

Volume II

1910

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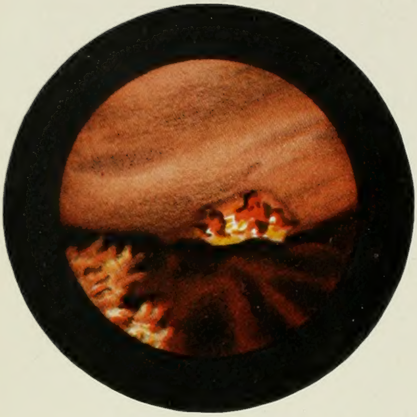
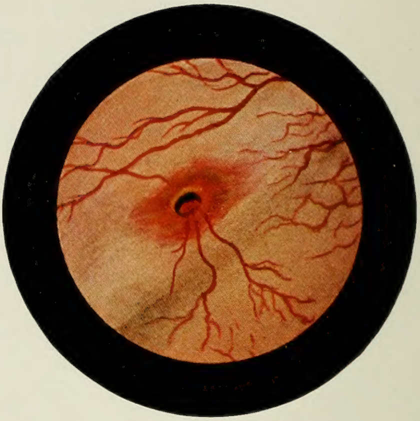


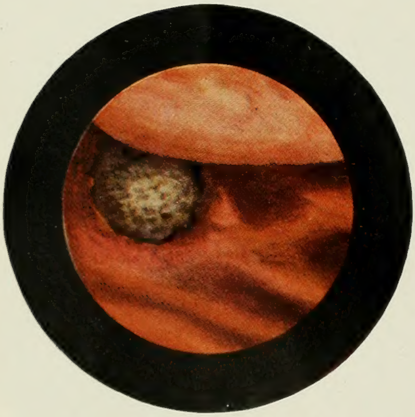
Carcinoma of Prostate and Bladder
See page 70

II



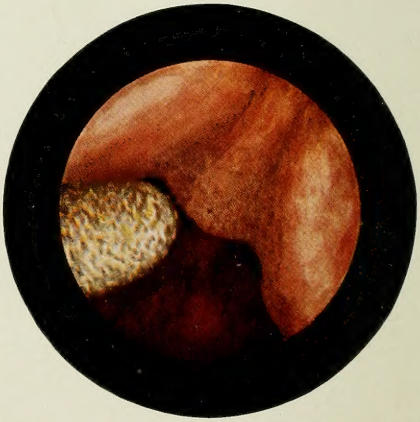
Ureteral Orifice with Calculus Impacted in the Vesical Portion of the Ureter. *See page* 71

III



Vesical Calculus Occupying a Sacculation Posterior to the Prostate. *See page* 72

IV



Vesical Calculus Resting on Hypertrophied Prostate. *See page* 72

CYSTOSCOPY AS AN AID TO DIAGNOSIS.

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Surgical Service

SURGICAL STATISTICS FOR 1910

ALIMENTARY SYSTEM INTESTINES	Operations	Cured	Improved	Unimproved	Died	Totals
Colitis	...	1	1	1	...	3
Colitis (ulcerative)	...	...	1	...	...	1
Colitis (ulc., hemorrhage)	...	...	...	...	1	1
Constipation	...	2	1	...	...	3
Duodenal ulcer	4	2	2	1	1	6
Enteritis	...	1	...	...	...	1
Enterolith, dementia	...	...	...	...	1	1
Enteroptosis, gastritis	...	...	...	1	...	1
Gastro-enteritis	...	1	...	...	...	1
Ileus	8	5	2	...	2	9
Ileus, carcinoma of intestines	4	1	1	...	2	4
Ileus, fibromyoma uterus, salp-oophoritis	1	...	...	...	1	1
Ileus, salpingitis	1	...	...	...	1	1
Ileus (paralytic), diverticulitis	1	...	...	...	1	1
Intestinal perforation, gen. peritonitis	1	...	...	...	1	1
Intussusception	3	4	...	...	...	4
	23	17	8	3	11	39
HERNIA						
Hernia (femoral)	18	18	...	...	...	18
Hernia (fem., strangulated)	7	5	...	...	2	7
Hernia (inguinal)	117	117	...	8	...	125
Hernia (ing.), del. tremens	...	...	...	1	...	1
Hernia (ing.), pul. embolus	1	...	...	...	1	1
Hernia (ing.), pul. thrombosis	1	...	...	...	1	1
Hernia (ing.), tbc. peritonitis, pul. tbc.	...	...	...	1	...	1
Hernia (ing. obstructed)	1	1	...	...	...	1
Hernia (ing. strangulated)	7	6	...	...	1	7
Hernia (ing. strangulated), myocarditis	1	...	...	...	1	1
Hernia (umbilical)	5	5	...	1	...	6
Hernia (ventral)	15	16	...	3	...	19
Hernia (ventral), ileus	1	1	...	1	...	1
Hernia (vent. strangulated)	1	...	...	...	1	1
Hernia (vent. strang.), ileus, gangrene of gut	1	...	...	...	...	1
	176	169	...	15	7	191
LIVER						
Abscess of liver	1	1	...	...	...	1
Abs. of liver, fecal abs	1	...	...	...	1	1
Abs. of liver, pul. tbc.	1	...	1	...	...	1
Cirrhosis (atrophic)	...	...	...	1	...	1
Cyst of liver, adhesions	...	...	...	1	...	1
Hepatoptosis	...	...	1	...	...	1
	3	1	2	2	1	6
BILE PASSAGES						
Calculus in common duct	1	1	...	...	...	1
Cholangitis (catarrhal)	...	...	1	...	...	1
Cholecystitis	3	3	2	1	...	6
Cholecystitis, adhesions	1	1	...	...	...	1

ALIMENTARY SYSTEM BILE PASSAGES	Operations	Cured	Improved	Unimproved	Died	Totals
Cholecystitis (supp.)	1	1	...	...	...	1
Cholelithiasis	29	27	1	5	1	34
Cholelithiasis, rupture of gall bladder	1	...	1	...	...	1
Cholelithiasis, pneumonia	1	...	...	...	1	1
Cholelithiasis, cholangitis	1	1	...	...	...	1
Fistula in gall bladder	1	1	...	...	...	1
	39	35	5	6	2	48
MOUTH, GUMS AND TEETH						
Alveolar abscess	3	4	...	...	...	4
Alveolar abs., septicemia	1	...	...	...	1	1
	4	4	...	...	1	5
OESOPHAGUS						
Stricture of oesophagus	1	...	2	...	...	2
PERITONEUM						
Adhesions	9	8	4	...	...	12
Cyst of mesentery	2	2	...	...	...	2
Peritonitis, cause unknown	1	...	...	...	1	1
Pelvic cellulitis	...	1	...	...	...	1
Peritoneal abs. (retro-cecal)	1	1	...	...	...	1
Retro-peritoneal abs.	3	3	...	...	...	3
	16	15	4	...	1	20
PHARYNX, TONSILS AND NASOPHARYNX						
Abscess (peritonsillar)	3	1	1	1	...	3
Abscess (retropharyngeal)	2	1	...	...	1	2
Adenoids	19	19	...	...	...	19
Hyp. tonsils	17	17	...	1	...	18
Hyp. tonsils, adenoids	121	117	1	3	...	121
Hyp. tonsils, bronchitis	...	...	...	1	...	1
Submerged tonsil	1	1	...	...	...	1
Tonsillar hemorrhage	...	1	...	...	...	1
Tonsillitis	...	2	...	1	...	3
	163	159	2	7	1	169
RECTUM						
Fissure in ano	6	4	3	1	...	8
Fistula in ano	34	33	4	...	...	37
Fistula in ano, pul. tbc.	1	...	1	1	...	2
Fistula in ano, specific urethritis	1	...	1	...	...	1
Fistula (fecal)	4	4	1	...	...	5
Hemorrhoids	88	84	5	5	...	94
Ischio-rectal abscess	18	15	5	1	...	21
Ischio-rectal abs., vaginitis	1	1	...	...	...	1
Perirectal abscess	8	5	3	...	...	8
Proctitis	...	...	1	...	...	1
Prolapse of rectum	4	2	1	...	1	4

	Op.	C.	Imp.	Un.	D.	Tot.
Rectum—Cont.						
Rectocele	2	2	...	...	...	2
Stricture of rectum	3	2	1	...	...	3
	170	152	26	8	1	187
STOMACH						
Contraction of stomach	1	...	1	...	...	1
Gastritis (chr.)	...	1	2	1	...	4
Gastritis, hypermetropia	...	...	1	...	...	1
Hyperchlorhydria	...	...	1	...	...	1
Obstruction of pylorus	1	1	...	...	...	1
Stenosis of pylorus	1	...	...	...	1	1
Stenosis of pylorus, pneumonia	1	...	...	...	1	1
Ulcer (gastric)	2	3	1	...	...	4
Ulcer (gastric), supp. pleurisy	1	...	...	...	1	1
	7	5	6	1	3	15
VERMIFORM APPENDIX						
Appendicitis (acute)	42	42	...	...	...	42
Appendicitis (ac. with peritonitis)	21	14	...	...	7	21
Appendicitis (ac. with peritoneal abscess)	38	35	1	...	2	38
Appendicitis (ac. with per. abs.) pneumonia	1	...	...	...	1	1
Appendicitis (ac. with per. abs.) ulceration of gut	1	...	...	...	1	1
Appendicitis (ac. with per. abs.), fibro-purulent peritonitis	1	...	...	...	1	1
Appendicitis (ac. catarrhal)	6	6	...	...	...	6
Appendicitis (subac. cat.)	12	12	...	...	...	12
Appendicitis (chr. cat.)	5	5	...	...	...	5
Appendicitis (chronic)	105	104	3	1	...	108
Appendicitis (chr.) parotitis	1	1	...	...	...	1
Appendicitis (chr. with adhesions)	2	2	...	...	...	2
Appendicitis (gangrenous)	11	11	...	...	...	11
Appendicitis (gang. with peritonitis	13	8	1	...	4	13
Appendicitis (perforative with peritonitis)	5	4	...	...	1	5
Appendicitis (sub-acute)	32	33	1	...	...	34
Appendicitis (suppurative)	20	20	...	...	...	20
Hydrops of appendix	1	1	...	...	...	1
	317	298	6	1	17	322
CARDIO-VASCULAR SYSTEM						
BLOOD						
Myelogenous leukemia	1	...	...	...	1	1
ARTERIES						
Thrombosis (cerebral)	...	...	...	...	1	1
Thrombosis (femoral)	1	...	...	...	1	1
	1	...	...	...	2	2
VEINS						
Phlebitis of arm	1	1	...	...	...	1
Phlebitis of leg	1	...	3	...	...	3
Phlebitis of thigh, nephritis	1	...	...	...	1	1
Thrombo-phlebitis of finger	1	1	...	...	...	1
Varicose veins of leg	17	17	...	2	...	19
Var. veins with phlebitis	1	1	...	...	...	1
Var. veins, pregnancy	...	...	...	1	...	1
	22	20	3	3	1	27

	Op.	C.	Imp.	Un.	D.	Tot.
HEART						
Endocarditis (acute)	...	...	...	1	...	1
Endocarditis (chronic)	...	...	1	...	...	1
Endocarditis (septic)	...	...	...	1	1	2
Pericarditis	...	...	1	...	...	1
	...	...	2	2	1	5
LYMPH GLANDS						
Lymphadenitis (axillary)	...	...	1	...	...	1
Lymphadenitis (cervical)	8	9	2	...	...	11
Lymphadenitis (inguinal)	10	8	3	...	...	11
	18	17	6	...	...	23
SPLEEN						
Perisplenitis	...	...	1	...	...	1
CONNECTIVE TISSUE						
Abscess of arm	1	...	1	...	...	1
Abscess of axilla	1	1	...	...	...	1
Abscess of back	1	1	...	...	...	1
Abscess of breast	2	2	...	...	...	2
Abscess of face	4	3	1	...	...	4
Abscess of knee	1	1	...	...	...	1
Abscess of neck	8	5	3	...	...	8
Abscess of popliteal space	1	1	...	...	...	1
Abscess of scalp	1	1	...	...	...	1
Abscess of thigh	2	1	1	...	...	2
Abscess in appendectomy scar	1	1	...	...	...	1
Cellulitis of arm	5	5	...	...	...	5
Cellulitis of arm, uremia	1	...	...	...	1	1
Cellulitis of chest wall, senility	1	...	...	...	1	1
Cellulitis of face	1	...	1	...	...	1
Cellulitis of foot	3	4	1	...	...	5
Cellulitis of hand	20	15	6	...	...	21
Cellulitis of leg	3	3	...	...	...	3
Cellulitis of neck	2	2	...	...	...	2
Cicatricial contracture of mouth	1	...	1	...	...	1
Emphysema of neck	...	...	...	...	1	1
Hematoma of buttocks	1	...	...	...	1	1
Hematoma of scalp	1	1	...	...	...	1
Oedema of scrotum	...	1	...	...	...	1
	62	48	15	...	4	67
DUCTLESS GLANDS						
Goitre (aneurysmal)	...	...	...	1	...	1
Goitre (simple)	4	3	2	2	...	7
Goitre (simple), pregnancy	...	...	1	...	...	1
Goitre (exophthalmic)	2	...	1	2	1	4
Thyroid cyst	1	1	...	...	...	1
Thyro-glossal cyst	1	1	...	...	...	1
	8	5	4	5	1	15
MUSCULAR SYSTEM						
Bursitis of elbow	1	1	...	...	...	1
Bursitis—olecranon	2	2	...	...	...	2
Bursitis—prepatellar	1	1	...	...	...	1
Contracture of hand	4	2	2	...	...	4
Dupuytren's contraction	2	1	1	...	...	2
Muscular rheumatism	1	1	...	...	...	1
Muscular torticollis	1	1	...	...	...	1
Myositis (lumbar)	...	...	...	1	...	1
Psoas abscess	1	1	...	...	...	1
	13	10	3	1	...	14
NERVOUS SYSTEM						
BRAIN						
Degeneration of cerebrum	1	...	...	...	1	1
Hemiplegia, muscular atrophy	...	...	...	1	...	1
	1	...	...	1	1	2

	Op.	C.	Imp.	Un.	D.	Tot.
DISEASES OF THE MIND						
Genl. paresis of the insane	...	...	...	1	...	1
NERVES						
Neuralgia (brachial)	2	2	...	...	...	2
Neuralgia (sacral)	...	...	1	...	...	1
Neuralgia (trifacial)	3	2	1	...	...	3
Neuralgia (trifacial), cerebral oedema	1	...	...	...	1	1
Neuritis (peripheral)	1	...	1	...	...	1
	7	4	3	...	1	8
NERVOUS DISEASES OF UNKNOWN ORIGIN						
Catalepsy	...	...	...	1	...	1
Chorea	...	...	...	1	...	1
Epilepsy	2	...	1	3	...	4
Erythromelalgia	...	...	1	...	...	1
Neurasthenia	...	1	8	2	...	11
Spasmodic torticollis	...	...	1	...	...	1
	2	1	11	7	...	19
SPINAL CORD						
Anterior poliomyelitis	...	...	2	...	...	2
Paralysis, atrophy of arm	...	...	...	1	...	1
	...	...	2	1	...	3
OSSEOUS SYSTEM						
BONES						
Coccygodynia	2	2	...	...	...	2
Cyst, sacro-coccygeal	1	1	...	...	...	1
Hypertrophy of turbinate bones	1	1	1	...	...	2
Necrosis—alveolar process	1	...	1	...	...	1
Necrosis—inf. maxilla	3	1	2	...	...	3
Necrosis—sup. maxilla	1	1	...	...	...	1
Necrosis of phalanx	1	...	1	...	...	1
Osteitis—maxillary antrum	1	1	...	...	...	1
Osteomyelitis of femur	1	2	2	...	...	4
Osteomy. of femur, septicemia	1	...	...	...	1	1
Osteomy. of foot	9	7	2	1	...	10
Osteomy. of inf. maxilla	1	...	1	...	...	1
Osteomy. of mastoid bone	1	...	1	...	...	1
Osteomy. of malar bone	1	...	1	...	...	1
Osteomy. of parietal bone	1	...	1	...	...	1
Osteomy. of phalanx	1	1	...	...	...	1
Osteomy. of stump of foot	1	1	...	...	...	1
Osteomy. of stump of knee	1	1	...	...	...	1
Osteomy. of tibia	3	1	2	...	...	3
Periosteitis of tarsus	...	1	...	...	...	1
Subperiosteal abcess of tibia	1	1	...	...	...	1
	33	22	15	1	1	39
JOINTS						
Ankylosis of Elbow	1	...	1	...	...	1
Arthritis (chr.) of knee	1	...	2	...	...	2
Arth. (supp.) of knee	1	1	...	...	...	1
Arth. (supp.) of knee, pyemia	1	...	...	...	1	1
Hypertrophy of synovial folds of knee	3	3	1	...	...	4
Hysterical knee joint	...	...	1	...	...	1
Loose body in knee joint	2	2	1	...	...	3
Osteo-arthritis of hip	2	...	3	1	...	4
Rheumatism, chr. articular	...	...	...	1	...	1
Synovitis of hip	...	...	1	...	...	1
Synovitis of knee	...	3	2	...	...	5
	11	9	12	2	1	24

	Op.	C.	Imp.	Un.	D.	Tot.
REPRODUCTIVE SYSTEM						
FEMALE URETHRA						
Urethral calculus	...	...	1	...	...	1
Urethral caruncle	2	1	...	...	1	2
	2	1	1	...	1	3
OVARY						
Abscess, tubo-ovarian	9	9	...	...	...	9
Abscess (tubo-ov.), perforation into rectum	1	...	...	...	1	1
Abscess (tubo-ov.), residual abscess	2	...	...	...	2	2
Abscess (tubo-ov.), septic endometritis	...	...	1	...	...	1
Cystic ovary	37	36	...	1	...	37
Cystic ovary, multilocular	2	2	...	...	...	2
Cystic ovary, pregnancy	...	...	...	1	...	1
Oophoritis	2	3	...	...	...	3
	53	50	1	2	3	56
UTERINE TUBE						
Hematosalpinx	2	2	...	...	...	2
Hydrosalpinx	5	5	...	...	...	5
Pyosalpinx	25	23	1	2	...	26
Pyosalpinx with abscess	2	1	1	...	...	2
Pyosalpinx, septic metritis	1	...	...	...	1	1
Salpingitis (acute)	3	3	2	1	...	6
Salpingitis (chronic)	52	58	11	6	...	75
Salpingitis (chr. with peritonitis)	2	1	...	...	1	2
Salpingitis (chr.) pregnancy	...	1	...	1	...	2
Salpingitis (gangrenous)	1	1	...	...	...	1
Salpingitis (purulent)	2	2	...	...	...	2
Salpingo-oophoritis	18	20	1	...	...	21
	113	117	16	10	2	145
UTERUS						
Anteflexion	1	1	...	...	...	1
Cervicitis	1	1	...	...	...	1
Cyst (intra-ligamentous)	5	3	1	...	1	5
Cyst of cervix uteri	1	1	...	...	...	1
Dysmenorrhea	4	4	1	...	...	5
Endocervicitis (glandular)	1	1	...	...	...	1
Endometritis (acute)	2	2	...	...	...	2
Endometritis (chronic)	83	85	1	2	...	88
Endometritis (chr.), carcinoma of uterus	1	...	1	...	...	1
Endometritis (glandular)	1	1	...	...	...	1
Endometritis (hypertrophic)	2	2	...	...	...	2
Endometritis (septic)	4	4	1	...	...	5
Endometritis (septic with peritonitis)	1	...	...	...	1	1
Hypertrophy of cervix	3	3	...	...	...	3
Menorrhagia	1	1	...	...	...	1
Metrorrhagia	1	1	1	...	...	2
Prolapse of uterus	16	14	...	3	...	17
Prolapse, laceration of cervix and perineum	...	...	...	1	...	1
Retroversion	64	63	...	5	1	69
Stenosis of cervix uteri	3	2	1	...	...	3
	195	189	7	11	3	210
PREGNANCY, ETC.						
Abortion (complete)	...	2	...	...	...	2
Abortion (incomplete)	24	23	...	...	1	24
Abortion (threatened)	5	6	...	...	...	6
Abortion, hemorrhages	1	1	...	...	...	1
Abortion, streptococcic septicemia	1	...	...	...	1	1
Ectopic gestation	11	10	1	...	1	12

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Pregnancy, Etc.—Cont.						
Ectopic gestation, pelvic hematocele	2	1	1	...	...	2
Ectopic gestation, salpingitis	1	1	...	...	...	1
Pelvic abscess	11	10	1	...	...	11
Pregnancy	2	...	1	5	...	6
Pregnancy, pul. tbc.	...	...	1	...	...	1
Retained placenta	32	32	1	1	...	34
Vomiting of pregnancy	1	1	...	...	...	1
	91	87	6	6	3	102
VAGINA						
Prolapse ant. vaginal wall	5	5	...	...	...	5
Prolapse post. vaginal wall	4	4	1	...	...	5
Relaxed vaginal outlet	1	1	...	...	...	1
	10	10	1	...	...	11
VULVA						
Abscess—vulvo-vaginal	2	2	...	...	...	2
Abscess—Bartholin's gland	1	1	...	...	...	1
Cyst—Bartholin's gland	1	1	...	...	...	1
Cyst—vulvo-vaginal	1	1	...	...	...	1
Hematoma of vulva	1	1	...	...	...	1
Varicosities of vulva	1	...	1	...	...	1
	7	6	1	...	...	7
MALE URETHRA						
Abscess—periurethral	1	1	...	...	...	1
Abscess—periurethral, stricture	1	1	...	...	...	1
Fistula of urethra	1	1	...	...	...	1
Fibrous thickening of urethra	1	...	1	...	...	1
Obstruction of urethra	1	...	1	...	...	1
Stricture of urethra	14	13	2	2	...	17
	19	16	4	2	...	22
PENIS						
Gangrene of prepuce	...	...	1	...	...	1
Phimosis	9	9	...	...	...	9
Redundancy of prepuce	7	7	...	...	...	7
	16	16	1	...	...	17
PROSTATE						
Abscess of prostate	1	1	...	...	...	1
Abscess with uremia	1	...	...	...	1	1
Hypertrophy of prostate	15	11	7	2	4	24
Hypertrophy, dementia	...	...	...	1	...	1
Prostatis (acute)	...	1	...	...	...	1
	17	13	7	3	5	28
SPERMATIC CORD						
Hydrocele	17	16	1	...	...	17
Varicocele	25	25	...	1	...	26
	42	41	1	1	...	43
TESTICLE						
Epididymitis	...	1	...	...	...	1
Orchitis (acute)	1	...	...	...	...	1
	1	1	...	...	...	1
MAMMARY GLAND						
Mastitis	3	4	1	1	...	6
RESPIRATORY SYSTEM						
BRONCHI						
Bronchitis	1	...	...	...	...	1
Bronchitis (foetid)	...	...	1	...	...	1
	1	...	1	...	...	2

	Op.	C.	Imp.	Un.	D.	Tot.
LUNGS						
Abscess of lung	1	...	...	...	1	1
Emphysema (pulmonary)	...	...	...	1	...	1
Gangrene of lung	1	...	...	...	1	1
Pneumonia (broncho)	...	1	1	...	...	2
Pneumonia (lobar)	...	...	2	...	1	3
	2	1	3	1	3	8
PLEURAE						
Pleurisy (chronic)	...	...	...	1	...	1
Pleurisy with effusion	1	1	1	...	...	2
Pleurisy (suppurative)	18	13	2	...	4	19
Pleurisy (supp.) collapse of lung	1	...	...	1	...	1
	20	14	3	2	4	23
NASAL CAVITY						
Deviation of nasal septum	8	8	...	...	...	8
Epistaxis	...	1	...	...	...	1
Frontal sinusitis	1	1	...	...	...	1
Nasal obstruction	8	8	...	...	...	8
	17	18	...	...	...	18
SENSE ORGANS						
ORGAN OF HEARING						
Mastoiditis	15	13	2	1	1	17
Mastoiditis, encephalitis	1	...	...	...	1	1
Mastoiditis, meningitis	1	...	...	...	1	1
Mastoiditis, otitis media	...	...	1	...	...	1
Mastoiditis, pul. tbc.	3	2	...	...	2	4
Mastoiditis, septicemia	1	...	...	...	1	1
Mastoiditis (suppurative)	1	1	...	...	...	1
Otitis media	3	4	3	2	...	9
Otitis media, empyema of antrum of highmore	1	1	...	...	...	1
Otitis media (purulent)	1	1	...	...	...	1
	27	22	6	3	6	37
ORGAN OF VISION						
Cataract	9	7	5	...	...	12
Cataract, nephritis	...	...	...	1	...	1
Conjunctivitis	...	1	1	...	...	2
Entropion	1	...	1	...	...	1
Glaucoma	2	1	1	...	...	2
Irido-choroiditis	1	1	1	...	...	2
Irido-cyclitis	...	1	...	...	...	1
Iritis	1	3	...	...	...	3
Keratitis	1	...	1	...	...	1
Oedema of eyelids	1	...	1	...	...	1
Strabismus	3	3	...	...	...	3
	19	17	11	1	...	29
TEGUMENTARY SYSTEM						
Carbuncle of finger	1	1	...	...	...	1
Carbuncle of neck	3	2	...	...	1	3
Carbuncle of neck, brain abscess, pyemia	1	...	...	...	1	1
Eczema of leg	...	...	1	...	...	1
Furuncle of ear, eczema	...	...	1	...	...	1
Furunculosis	1	1	...	...	...	1
Epidermal cyst of sacral region	1	1	...	...	...	1
Pilonidal cyst	2	2	...	...	...	2
Purpura hemorrhagica	...	...	...	1	...	1
Sebaceous cyst of head	...	1	...	...	...	1
Sebaceous cyst of scrotum	1	1	...	...	...	1
Ulcer of foot from frost-bite	1	1	...	...	...	1
Ulcer of foot, senile gangrene	...	...	...	1	...	1
Ulcer of leg, eczema	...	1	...	...	...	1
Ulcer (perforative) of foot	...	...	1	...	...	1
Ulcers (varicose) of leg	...	...	...	1	...	1
	11	11	3	3	2	19

URINARY SYSTEM

KIDNEY

	Op.	C.	Imp.	Un.	D.	Tot.
Hydronephrosis	1	1	...	...	...	1
Nephritis (chr. interstitial)	...	1	1	1	...	3
Nephritis (suppurative)	1	...	...	...	1	1
Nephrolithiasis	...	...	2	...	...	2
Nephroptosis	2	2	...	...	...	2
Movable kidney	3	2	1	...	...	3
Movable kidney, pul. tbc.	...	...	1	...	...	1
Perinephritis	1	1	...	...	...	1
Pyelitis	...	...	1	...	...	1
Pyelitis (suppurative)	1	...	1	...	...	1
Pyelo-nephritis	1	1	...	...	...	1
Pyonephrosis	1	5	4	...	1	10
Renal calculus	1	2	1	2	...	5
Renal calculus, pyelonephritis with abscess	1	...	...	...	1	1
	13	15	12	3	3	33

URETER

	Op.	C.	Imp.	Un.	D.	Tot.
Calculus in ureter	3	3	...	1	1	5
Calculus, delirium tremens	...	...	...	1	...	1
Ureteral colic	...	2	1	2	...	5
Ureteritis	1	1	...	...	...	1
	4	6	1	4	1	12

URINARY BLADDER

	Op.	C.	Imp.	Un.	D.	Tot.
Calculus in bladder	3	3	...	...	...	3
Cystitis	...	4	2	...	...	6
Cystitis, tabes dorsalis	...	...	...	1	...	1
Retention of urine	1	1	2	...	...	3
	4	8	4	1	...	13

DISEASES DUE TO ANIMAL PARASITES

	Op.	C.	Imp.	Un.	D.	Tot.
Ascares lumbracoides	...	1	...	...	...	1
Scabies	...	...	...	1	...	1
Trichinosis	...	...	1	...	...	1
	...	1	1	1	...	3

CONGENITAL MALFORMATIONS

	Op.	C.	Imp.	Un.	D.	Tot.
Branchial genetic cyst	1	...	1	...	...	1
Cataract	1	...	1	...	...	1
Cleft palate	1	1	...	...	...	1
Contraction of foot	1	...	1	...	...	1
Fistula—recto-perineal	2	2	...	...	...	2
Fistula—recto-urethral	1	...	1	...	...	1
Fistula—recto-vaginal	2	2	...	1	1	4
Hare lip	2	2	...	...	...	2
Hydrocephalus	1	...	...	...	1	1
Infantile uterus	9	9	...	...	...	9
Maeckel's diverticulum	2	1	...	...	1	2
Malposition of kidney	1	1	...	...	...	1
Opacity of lens of eye	.	...	1	...	...	1
Pyloric spasm	...	...	1	...	...	1
Supernumerary toe	1	1	...	...	...	1
Tongue-tie	1	1	...	...	...	1
	26	20	6	1	3	30

DEFORMITIES

	Op.	C.	Imp.	Un.	D.	Tot.
Deformities of feet	1	...	1	...	...	1
Flap-ears	1	1	...	...	...	1
Flat nose from trauma	...	...	...	1	...	1
Hallux valgus	3	3	...	...	...	3
Hammer-toe	1	1	...	...	...	1
Pes planus	...	...	...	1	...	1
Talipes equinus	3	3	...	...	...	3
Webbed fingers	1	1	...	...	...	1
	10	9	1	2	...	12

LOCAL INJURIES

	Op.	C.	Imp.	Un.	D.	Tot.
Burns of arm and chest	1	...	1	...	...	1
Burns of back	...	1	...	...	...	1
Burns of chest	...	1	...	...	...	1
Burns of face	...	2	1	...	...	3
Burns of leg	...	1	...	...	...	1
Burns—extensive	...	...	...	...	1	1
Concussion of brain	1	3	...	...	...	3
Contusion of arm, shoulder	...	2	1	...	...	3
Contusion of face	...	1	1	...	...	2
Contusion of foot	...	1	1	...	...	2
Contusion of hand	...	1	...	...	...	1
Contusion of spine	...	...	2	...	...	2
Contusions—extensive	...	1	...	...	...	1
Dislocation of elbow	...	1	...	...	...	1
Dislocation of hip	1	...	...	...	1	1
Dislocation of meniscus	...	...	...	1	...	1
Dislocation of shoulder	...	1	...	...	...	1
Dislocation of vertebrae	...	...	...	...	1	1
Displacement of transverse colon	1	1	...	...	...	1
Division of tendons of wrist	1	1	...	...	...	1
Foreign body in face	1	1	...	...	...	1
Foreign body in nephrotomy sinus	1	1	...	...	...	1
Foreign body in oesophagus	2	2	...	...	...	2
Foreign body in skull	1	1	...	...	...	1
Foreign body in throat	1	1	...	...	...	1
Fracture of base of skull	...	2	1	...	3	6
Fracture of bones of foot	...	1	1	...	...	2
Fracture of clavicle	...	2	2	...	...	4
Fracture of clavicle (ununited)	...	...	...	1	...	1
Fracture of coccyx	.	1	...	...	...	1
Fracture of femur	...	4	2	...	...	6
Fracture of femur and tibia	1	1	...	...	...	1
Fracture of femur, oedema of lungs	...	...	...	...	1	1
Fracture of femur (spontaneous)	...	...	1	...	...	1
Fracture of femur (ununited)	2	2	...	1	...	3
Fracture of fibula	...	1	2	...	...	3
Fracture of fibula (Potts')	...	2	2	...	...	4
Fracture of humerus	3	5	4	...	...	9
Fracture of jaw	...	...	3	...	...	3
Fracture of jaw (ununited)	1	...	1	1	...	2
Fracture of neck of femur	...	2	1	...	...	3
Fracture of nose	...	1	...	...	...	1
Fracture of os calcis	...	...	3	...	...	3
Fracture of patella	4	6	1	...	...	7
Fracture of phalanx	...	1	...	...	...	1
Fracture of radius	1	1	...	...	...	1
Fracture of radius (Colles')	...	2	2	...	...	4
Fracture of radius (Colles') non-union	1	...	1	...	...	1
Fracture of radius and ulna	3	4	...	1	...	5
Fracture of scapula	...	...	1	...	...	1
Fracture of spine	...	...	...	1	...	1
Fracture of tibia	...	2	2	...	...	4
Fracture of tibia and fibula	...	2	...	...	...	2
Fracture of tibia (ununited)	...	1	...	...	...	1
Fracture of vault of skull	2	2	...	...	...	2
Fracture callus of rib	1	1	...	...	...	1
Fracture callus of thumb	...	...	1	...	...	1
Fracture separation of epiphysis of femur	...	1	...	...	...	1
Gangrene of finger	1	1	...	...	...	1
Gangrene of foot	1	1	...	...	...	1
Gangrene of foot, arteriosclerosis	1	...	...	...	1	1
Gangrene of gut, mesenteric thrombosis	1	...	...	...	1	1
Gangrene (dry) of toe	...	...	1	...	...	1
Laceration of cervix uteri	35	33	...	2	...	35
Laceration of pelvic floor	1	1	...	...	...	1

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Local Injuries—Cont.						
Laceration of perineum	76	75	1	9	...	85
Perforation of uterus	3	2	...	...	1	3
Rupture of kidney	1	1	...	...	...	1
Rupture of sphincter ani	1	1	...	...	...	1
Rupture of ureter	1	1	...	...	...	1
Rupture of urinary bladder	1	1	...	...	...	1
Sinus (persistent) of abdomen	...	1	1	...	1	3
Sinus (per.) of chest wall	...	...	1	...	...	1
Sinus (per.) of face	...	...	...	1	...	1
Sinus (per.) of leg	...	...	1	...	...	1
Sinus (per.) of neck	1	1	...	...	...	1
Sinus (per.) of thigh	2	2	...	...	...	2
Sprain of ankle	...	1	...	...	...	1
Sprain of knee	...	...	2	...	...	2
Tonsilectomy hemorrhage	...	1	...	...	...	1
Wound (gunshot) of face	...	...	...	1	...	1
Wound (gunshot) of knee	...	1	...	...	...	1
Wound (incised) of hand	1	1	...	...	...	1
Wound (lacerated) of nose	...	1	...	...	...	1
Wound (lac.) of scalp	1	3	1	...	...	4
Wound (lac.) of thigh	...	1	...	...	...	1
Wound in laparotomy scar	...	...	1	...	...	1
	158	201	47	19	11	278
DISEASES DUE TO MICRO-ORGANISMS						
INFECTIVE DISEASES						
Chancroid of groin	1	...	1	...	...	1
Dysentery (amoebic)	1	...	...	...	1	1
Erysipelas	...	1	...	...	...	1
Gonococcus arthritis of knee	...	1	...	...	...	1
Gonococcus epididymitis	...	1	1	...	...	2
Gonococcus proctitis	...	...	...	1	...	1
Gonococcus salpingitis	...	...	1	...	...	1
Gonococcus tenosyn. of foot	1	1	...	...	...	1
Gonococcus urethritis	...	1	...	...	...	1
Gonococcus vaginitis	...	...	1	...	...	1
Measles	...	...	...	...	1	1
Rheumatism (ac. articular)	...	...	1	...	...	1
Syphilis (secondary)	...	...	1	...	...	1
Syphilitic enteritis	...	...	...	1	...	1
Syph. gumma of buttock	...	...	...	1	...	1
Syph. gumma of leg	...	...	1	...	...	1
Syph. gumma of scrotum	1	...	1	...	...	1
Syph. gumma of sternum	...	...	1	...	...	1
Syph. hydrocele	1	1	...	...	...	1
Syph. osteitis of tibia	...	...	1	...	...	1
Syph. periosteitis of jaw	...	...	1	...	...	1
Syph. periosteitis of tibia	...	...	1	...	...	1
Syph. periproctitis	...	...	1	...	...	1
Syph. stenosis of larynx	1	...	1	...	...	1
Syph. ulcer of leg	...	...	1	...	...	1
Syph. ulcer of vulva	...	...	1	...	...	1
Tetanus from wound of foot	...	...	...	...	1	1
Tuberculosis of astragalus	1	...	1	...	...	1
Tbc. of bones of hand	1	...	1	...	...	1
Tbc. of carpal bones, septicemia	1	...	...	...	1	1
Tbc. of elbow	...	...	1	...	...	1
Tbc. of epididymis	1	...	1	...	...	1
Tbc. of femur	1	1	...	...	...	1
Tbc. of finger	1	...	1	...	...	1
Tbc. of glands of axilla	8	6	1	1	...	8
Tbc. of glands of groin	1	1	...	...	...	1
Tbc. of glands of neck	23	19	7	2	...	28
Tbc. of hand	1	1	...	...	...	1
Tbc. of hip	...	...	1	1	...	2
Tbc. of kidney	1	...	1	...	...	1
Tbc. of kidney and bladder	...	...	1	...	...	1
Tbc. of kidney, parotiditis	1	1	...	...	...	1
Tbc. of knee	2	2	2	1	...	5
Tbc. of lungs	...	...	...	1	...	1
Tbc. of peritoneum	2	2	3	...	1	6
Tbc. of pleura	...	...	1	...	1	2
Tbc. of spine (Potts')	...	...	1	2	...	3
Tbc. of testicle	3	3	...	...	...	3
Tbc. of uterine tubes	4	2	2	...	...	4
Tbc. of uterine tubes, peritonitis	3	.	2	...	1	3
Tuberculous meningitis	...	...	...	1	...	1
Tbc. pyelonephrosis	1	...	1	...	...	1
Tbc. pyonephrosis	1	...	1	...	...	1
Tbc. (miliary) of kidney and lungs	...	...	...	1	...	1
Typhoid fever	...	...	...	3	...	3
	63	44	45	16	7	112
LOCAL INFECTIONS						
Infection of arm	1	1	...	...	...	1
Infection of hand	6	6	...	...	...	6
Infection of herniotomy wound	1	1	...	...	...	1
Infection of toe	...	...	1	...	...	1
Stitch abscess	1	1	...	...	...	1
	9	9	1	...	...	10
NEOPLASMS						
Adenoma of breast	3	3	...	...	...	3
Adeno-carcinoma of ovary	2	1	1	...	...	2
Adeno-carcinoma of rectum	1	1	1	...	...	2
Adeno-fibroma of breast	10	10	1	...	...	11
Carcinoma of bladder	2	...	2	2	1	5
Carcinoma of breast	37	24	14	2	...	40
Carcinoma of cervix uteri	3	2	1	...	...	3
Carcinoma of chest wall	...	...	...	1	...	1
Carcinoma of ear	1	...	1	...	...	1
Carcinoma of face	...	...	...	2	...	2
Carcinoma of glands of neck	5	...	4	2	...	6
Carcinoma of groin	...	...	...	1	...	1
Carcinoma of inf. maxilla	1	1	...	...	...	1
Carcinoma of intestines	12	2	1	5	4	12
Carcinoma of liver	...	...	...	3	1	4
Carcinoma of neck	...	...	...	1	...	1
Carcinoma of oesophagus	...	...	1	...	...	1
Carcinoma of orbit	1	...	...	1	...	1
Carcinoma of ovary	2	...	1	...	1	2
Carcinoma of parotid gland	...	...	...	1	...	1
Carcinoma of pancreas	1	...	...	...	1	1
Carcinoma of peritoneum	1	...	...	...	1	1
Carcinoma of prostate	...	...	...	1	...	1
Carcinoma of rectum	4	1	1	3	3	8
Carcinoma of stomach	10	1	4	6	3	14
Carcinoma of submental region	...	...	1	1	...	2
Carcinoma of testicle	1	...	1	...	...	1
Carcinoma of tongue	...	...	1	1	...	2
Carcinoma of urethra	1	1	...	...	...	1
Carcinoma of uterus	7	1	5	1	1	8
Carcinoma of vagina	4	1	3	1	...	5
Carcinomatosis	1	...	...	2	...	2
Condyloma acumenata	1	1	...	...	...	1
Cyst-adenoma of breast	3	3	...	...	...	3
Cyst-adenoma of ovary	1	...	1	...	...	1
Dermoid cyst of orbit	1	1	...	...	...	1
Dermoid cyst of ovary	2	2	...	...	...	2
Dermoid cyst of sacrum	1	1	...	...	...	1
Epithelioma of back	1	1	...	...	...	1
Epithelioma of cervix uteri	...	1	...	...	...	1
Epithelioma of conjunctiva	...	...	...	1	...	1
Epithelioma of face	15	8	6	3	...	17
Epithelioma of forehead	2	2	...	...	...	2
Epithelioma of frontal region	...	1	...	...	...	1
Epithelioma of neck	4	2	2	2	...	6
Epithelioma of orbit	1	1	...	1	...	2
Epithelioma of shoulder	1	1	...	...	...	1
Epithelioma, submental region	...	...	...	1	...	1
Epithelioma of vulva	1	1	1	...	...	6
Epulis	1	1	...	...	...	1
Exostosis of knee joint	1	...	1	...	...	1

	Op.	C.	Imp.	Un.	D.	Tot.
Neoplasms—Cont.						
Exostosis of scapula	1	1	...	...	...	1
Exostosis of skull	1	1	...	...	...	1
Exostosis of tibia	1	1	...	...	...	1
Fibroma of breast	1	1	...	...	...	1
Fibroma of cheek	1	...	1	...	...	1
Fibromyoma of uterus	76	74	3	5	1	83
Fibromyoma of uterus, dilatation of stomach	1	...	...	...	1	1
Fibromyoma of uterus, peritonitis, septicemia	1	...	...	...	1	1
Fibro-sarcoma of abdominal wall	1	1	...	...	...	1
Galactocele of breast	1	1	...	...	...	1
Glioma of occipital bone	1	...	1	...	...	1
Hypernephroma of kidney	2	...	1	1	1	3
Lipoma of abdominal wall	1	1	...	...	...	1
Lipoma of back	...	...	...	2	...	2
Lipoma of breast	2	2	...	...	...	2
Lipoma of buttock	1	1	...	...	...	1
Lipoma of neck	2	2	...	...	...	2
Lipoma of thigh	...	...	...	1	...	1
Lymphangioma of arm	...	...	...	1	...	1
Lymphangioma of neck	...	...	...	1	...	1
Lymphangioma of thigh	1	1	...	...	...	1
Lymphosarcoma of breast	...	...	...	...	1	1
Lymphosarcoma of neck	1	1	...	...	...	1
Lymphosarcoma of submental glands	1	...	1	...	...	1
Lupus of head	1	1	...	...	...	1
Myxoma of sacral region	1	...	1	...	...	1
New growth of bladder	...	...	...	1	...	1
New growth of colon	1	...	1	...	...	1
New growth of liver	...	...	1	...	...	1
New growth of intestines	1	1	...	...	...	1
New growth of ovary	1	...	...	1	1	2
New growth of pancreas	1	...	...	...	1	1
New growth of pylorus	1	...	1	...	...	1
New growth of sup. maxilla	2	1	...	...	1	2
Osteochondroma of foot	1	1	...	...	...	1
Osteosarcoma of sacrum	...	...	...	1	...	1
Osteosarcoma of sternum	...	...	...	1	...	1
Osteosarcoma of tibia	1	...	1	...	...	1
Papilloma of bladder	...	...	...	...	1	1
Papilloma of larynx	1	1	1	1	...	3
Papilloma of penis	1	1	...	...	...	1
Papilloma of uterus	1	1	1	...	...	2
Pedunculated fibroma, uterus	1	1	...	...	...	1
Pelvic hematocele	1	1	...	...	...	1
Polyp of ear	1	1	...	...	...	1
Polyp of uterus	6	6	...	...	...	6
Sarcoma of axilla	2	.	2	...	...	2
Sarcoma of breast	1	...	1	...	...	1
Sarcoma of chest wall	1	...	1	...	...	1
Sarcoma of eye	2	2	...	...	...	2
Sarcoma of inf. maxilla	3	...	3	...	...	3
Sarcoma of kidney	1	...	...	...	1	1
Sarcoma of leg	...	...	...	2	...	2
Sarcoma of liver	1	...	...	2	...	2
Sarcoma of nasal cavity	1	...	1	...	...	1
Sarcoma of neck	1	1	...	1	...	2
Sarcoma of orbit	1	...	1	...	...	1
Sarcoma of ovary	1	...	...	1	...	1
Sarcoma of popliteal space	1	...	1	...	...	1
Sarcoma of sup. maxilla	4	...	3	2	...	5
Sarcoma of tibia	...	...	...	1	...	1
Sarcoma of tonsil, pharynx	1	...	...	...	1	1
Sarcoma of uterus	1	...	1	...	...	1
Tumor of abdomen	...	...	...	1	...	1
Tumor of breast	3	3	...	1	...	4
Tumor of cerebrum	1	...	...	1	1	2
Tumor of kidney	...	...	...	1	...	1
Tumor of liver	...	...	...	1	...	1
Tumor of mediastinum	...	...	1	...	...	1
Tumor of ovary	1	1	...	...	...	1
Villous growth of bladder	...	...	...	1	...	1
	195	186	85	75	28	374
INTOXICATIONS						
Alcoholism	...	1	1	...	...	2
Diabetes mellitus	...	...	1	1	...	2
Diabetes, gangrene of leg	2	...	1	...	2	3
Diabetes, intestinal paralysis	1	...	...	...	1	1
Diabetes, renal calculus	...	...	1	...	...	1
	3	1	4	1	3	9
MISCELLANEOUS						
Donor in transfusion	...	...	1	...	...	1
For observation	...	1	...	...	...	1
Malingering	...	1	...	...	...	1
No pathological condition	...	...	...	28	...	28
	...	2	1	28	...	31

SUMMARY

	Op.	C.	Imp.	Un.	D.	Tot.
Alimentary System	920	855	61	43	45	1004
Cardio-Vascular System	42	37	12	5	5	59
Connective Tissue	62	48	15	....	4	67
Ductless Glands	8	5	4	5	1	15
Muscular System	13	10	3	1	....	14
Nervous System	10	5	16	10	2	33
Osseous System	44	31	27	3	2	63
Reproductive System	566	547	46	35	17	645
(Mammary Gland)	3	4	1	1	....	6
Respiratory System	40	33	7	3	7	51
Sense Organs	46	39	17	4	6	66
Tegumentary System	11	11	3	3	2	19
Urinary System	21	29	17	8	4	58
Animal Parasites	....	1	1	1	....	3
Congenital Malformations	26	20	6	1	3	30
Deformities	10	9	1	2	....	12
Local Injuries	158	201	47	19	11	278
Micro-organic Diseases	72	53	46	16	7	122
Neoplasms	195	186	85	75	28	374
Intoxications	3	1	4	1	3	9
Miscellaneous	....	2	1	28	....	31
TOTAL ... Cases Admitted: 2760	2250	2127	420	264	147	2958

ALIMENTARY SYSTEM

INTESTINES

Cecostomy	1
Colostomy	10
Entero-colostomy	5
Intestinal anastomosis	3
Resection of intestines	16

HERNIA

Femoral hernia repair	26
Inguinal hernia repair	128
Umbilical hernia repair	5
Ventral hernia repair	16

LIVER AND BILE PASSAGES

Cholecystenterostomy	1
Cholecystectomy	17
Cholecystostomy	6
Cholecystotomy	15
Choledochotomy	3
Hepatotomy	1
Incision for abscess of liver	1

MOUTH, TONGUE AND TEETH

Excision of thyro-glossal cyst	1
Extraction of lower incisors	1
Incision for alveolar abscess	4
Partial glossectomy	1

OESOPHAGUS

Dilatation of oesophagus	1
Removal of foreign body	2

PERITONEUM, OMENTUM AND RETRO-PERITONEAL TISSUE

Celiotomy	4
Division of adhesions	15
Exploratory celiotomy	33
Fixation of omentum	1

PHARYNX, TONSILS AND NASOPHARYNX

Adenoidectomy	17
Adenoidectomy and tonsilectomy	123
Incision for peritonsillar abscess	3
Incision for retropharyngeal abscess	2
Tonsilectomy	16

RECTUM, ANUS AND PERI-RECTAL TISSUES

Clamp and cautery	50
Cauterization of rectum	1
Dilatation of sphincter ani	6
Dilatation of stricture	3
Excision of fistula in ano	10
Excision of mucous membrane of rectum	7
Incision of fistula in ano	19
Incision of ischio-rectal abscess	17
Incision of peri-rectal abscess	8
Ligation of hemorrhoids	31
Proctectomy	2
Proctotomy	3

STOMACH

Gastrectomy (partial)	2
Gastro-enterostomy	13
Gastro-gastrostomy	1
Gastroplasty	1
Gastrostomy	3

VERMIFORM APPENDIX

Appendicectomy	215
Appendicectomy with drain	97
Appendicostomy	1
Drainage of appendicular abscess	7

CARDIO-VASCULAR SYSTEM

ARTERIES

Ligation of artery	1

VEINS

Ligation of vein	1
Phlebectomy	33
Phlebotomy	4

LYMPH GLANDS

Incision	2
Lymphadenectomy	25
Lymphadenectomy (tbc. cervical)	24
Splenectomy	1

CONNECTIVE TISSUE

Incision for abscess	27
Incision for cellulitis	36

DUCTLESS GLANDS

Excision of goitre	2
Excision of thyroid cyst	1
Thyroidectomy	3

MUSCULAR SYSTEM

Excision of bursae	5
Incision for psoas abscess	1
Myectomy	1
Removal of loose body in knee	2
Tendon transplantation	1
Tenoplasty	1
Tenorrhaphy	3
Tenotomy	7

NERVOUS SYSTEM

BRAIN

Decompression	2
Elevation of depressed fragments	1
Exploratory craniotomy	9
Paracentesis ventriculi	1
Subdural drainage	1

NERVES

Neurectomy	6

SPINAL CORD

Laminectomy	2

OSSEOUS SYSTEM

BONES

Excision of sacro-coccygeal cyst	1
Ostectomy	9
Osteotomy	10
Osteotomy with drain	4
Reduction (closed) of fracture	33
Reduction (open) of fracture	11
Resection of coccyx	2
Resection of rib	8

JOINTS

Arthrectomy	1
Arthrotomy	3
Excision of synovial folds	3
Removal of foreign body	1
Resection of capsule of tuberculous knee	1

REPRODUCTIVE SYSTEM

OVARY

Excision of cyst	9
Oophorectomy	27
Plastic on ovary	7

UTERINE TUBE

Incision for tuberculous salpingitis	1
Salpingectomy	52
Salpingo-oophorectomy	126

UTERUS

Curetage	165
Dilatation of cervix	8
Excision of intraligamentous cyst	5
Hysterectomy—(complete)	8
Hysterectomy (partial)	20
Hysterectomy (supravaginal)	51
Hysteropexy (round ligament)	33
Hysteropexy (ventral)	42
Hysterorrhaphy	3
Induction of abortion	9
Myomectomy	25
Stomatoplasty	7
Trachelectomy	8
Tracheloplasty	7
Trachelorrhaphy	24

VAGINA AND PELVIC FLOOR

Colpoplasty	2
Colporrhaphy	9
Colpotomy	14
Excision of cyst	2
Incision of cyst	2
Perineoplasty	6
Perineorrhaphy	83

URETHRA

Dilatation of urethra	3
Excision of urethral caruncle	2
Incision of periurethral abscess	2
Urethrotomy	16

PENIS

Amputation of penis	1
Circumcision	17

PROSTATE

Incision for abscess of prostate	1
Prostatectomy (perineal)	10
Prostatectomy (suprapubic)	5

TESTICLES

Orchidectomy	5
Transplantation of testicle	5

SPERMATIC CORD

Bottle op. for hydrocele	3
Excision of hydrocele sac	13

MAMMARY GLAND

Mastectomy (complete)	36
Mastectomy (partial)	4
Mastotomy	1

RESPIRATORY SYSTEM

LARYNX, BRONCHI AND TRACHEA

Laryngotomy	1
Removal of foreign body in bronchi	1

LUNGS AND PLEURAE

Thoracentesis	1
Thoracotomy	14

NASAL CAVITY

Plugging of nares	1
Submucous resection	16

ORGAN OF HEARING

Mastoidectomy (partial)	16
Mastoidotomy	6
Paracentesis	4

ORGAN OF VISION

Enucleation of eyeball	3
Excision of cataract	4
Needling for cataract	3
Plastic on eye	2

TEGUMENTARY SYSTEM

Excision of carbuncle	5
Excision of epidermal cyst	1
Excision of sebaceous cyst	1
Removal of foreign body	2
Skin graft	3

URINARY SYSTEM

KIDNEY

Incision for perinephritic abscess	2
Nephrectomy	12
Nephropexy	2
Nephrorrhaphy	2
Nephrotomy	3
Ureterotomy	1

BLADDER

Cystorrhaphy	1
Cystoscopy	6
Cystostomy	1
Cystotomy	9

DEFORMITIES AND CONGENITAL MALFORMATIONS

Excision of branchial genetic cyst	1
Excision of Maeckel's diverticulum	2
Freeing short frenum of tongue	1
Plastic repair on deformities	6
Plastic repair on cleft palate	1
Plastic repair on hare lip	2

INJURIES

Suture of wound	2

DISEASES DUE TO MICRO-ORGANISMS

Incision of local infection	12

NEOPLASMS

Excision	101
Plastic on regions involved	7

AMPUTATIONS

Amputation at elbow	1
Amputation of finger	8
Amputation through upper thigh	1
Amputation through middle thigh	5
Amputation through lower thigh	2
Amputation at knee	1
Amputation through upper leg	1
Amputation through middle leg	1
Amputation through lower leg	1
Amputation through metatarsus	2
Amputation of toe	7

MISCELLANEOUS

Radium treatment	25

NOTA BENE. As the Nomenclature principally used in tabulating these statistics was not adopted until late in the year, it has been impossible to secure entire exactitude and uniformity.—REGISTRAR.

RESECTION OF POSTERIOR ROOTS OF SPINAL NERVES WITHIN THE DURA, TO RELIEVE PAIN, PAIN REFLEX, ATHETOSIS AND SPASTIC PARALYSIS. (DANA'S OPERATION.)

ROBERT ABBE, M.D.

It is more than twenty-two years since Dr. Charles A. Dana asked me if I were willing to perform for him, at St. Luke's Hospital, an operation which had not heretofore been done and which he believed offered relief to a patient then suffering violent reflex neuralgic pains of the right arm, with muscle contraction associated with the spasms of pain, who already had had nerve stretching and amputation at the upper humerus by Dr. Bull with no abatement of pain. He conceived that the operation of "cutting the posterior roots within the dura—would cause ascending degeneration and destroy the sensory tract—even into the spinal cord."

The plans for this unique operation were made in Sept. 1888, as shown by Dr. Dana's letter to me. The operation was delayed by the patient's desire to have a neuroma of his stump resected first. With every precaution I operated at St. Luke's Hospital, Dec. 31st, 1888, assisted by Dr. Bangs and in the presence of a number of distinguished neurologists and surgeons—performing Dana's operation.

A full description was reported by me to the N. Y. State Med. Society, Feb. 6th, 1889, and published in its annals Feb., 1889.

It was the forerunner of two others done by me at St. Luke's under the same inspiration and published at length in a paper on the subject which I read in Boston and published in the Boston Medical and Surgical Journal, Oct. 1st, 1896, with a review of operations by others up to that date, a total of seven.

The fact that my three patients lived under observation for many years, and that I can contribute two recent cases of much interest, added to the wide attention now aroused which has caused many operators to resort to it; cause me to make a short review of this now accepted operation.

Its legitimate scope,—time and experience only will demonstrate. Pioneer work must be sifted of the cases which were imperfectly or wrongly done—while surgeons were feeling their way carefully along a new path. Few had dared to open the spinal dura mater, release the arachnoid fluid and touch the cord itself up to the date proposed by Dr. Dana.

It had always been known that to relieve neuralgia by resecting nerves—as in the cervical plexus—would paralyze the motor function beyond repair, hence the substitution of the more inefficient nerve stretching had been made.

It had been demonstrated that the posterior roots were the sensory carriers—and that nerve division eventuated in ascending degeneration.

Following the progressive boldness in opening the cerebral and spinal cavities, it was natural that some thinking student should propose just the operation devised by Dr. Dana.

The first publication of our cases was Feb. 9, 1889. This was the first published case in literature. On April 23rd following, there appeared a description of a case of persistent neuralgia of the leg, for which amputation had been done without benefit, in which Dr. Bennett, of London, resected the posterior roots of the four lower lumbar and one upper sacral roots intradurally. This was done seven days prior to my first case but three months after Dr. Dana had proposed it, and of which there had been much open discussion. During this period, the patient operated in London was subjected to amputation of his leg, in hope of relief, and failing that, was then suspected of syphilis and during a month treated by mercurials, with no record of any idea of this operation. Failing in all relief, the operation that had been under discussion at St. Luke's by Dr. Dana was done by Dr. Bennett. The patient was relieved of pain but died on the twelfth day, of cerebral apoplexy.

Much has been written about the technical methods of operating. It has been thought wisest by some to operate in two stages, first uncovering the dura and packing the wound open, then, after two days, slitting up the dura and dividing the roots. This has been thought desirable to avoid shock. This was done in my first case while under ether. Some of the roots external to the dura were divided after identification by electric stimulation, but as the pain was not altered after two days, a series of roots within the dura was divided with the patient not anaesthetized, but lying face downward. Opening the dura gave no pain, but handling the roots on a hook gave pain sense to the patient exactly corresponding to that felt before operation, notably in the eighth cervical, which would supply the pain.

All my other operations have been done at one session, and I prefer this latter, unless the patient's condition demands delay.

As now conducted the accepted method is to separate the muscles from the laminae on one side the spinous processes under an incision alongside the spines; then, to gnaw away the laminae by rongeurs. This is the method first proposed by me after much work on spinal cases for tumors, pressure of fracture, etc., in 1888. One valuable modification has been made by Taylor, namely, to limit the removal of the bone to one half lamina. This exposes the roots sufficiently for this operation and preserves the support and protection from pressure of the other half lamina and spinous process. I have found this adequate in my last case, and consider this technique now about perfect.

The operation is neither a bloody one nor tedious. The essentials are: Horizontal posture of the patient, three quarters turned upon his chest; a sharp pair of medium small rongeurs bent partly on the flat, and preferably with a "Gothic arch" cutting edge, such as I had made for me by Charrier in Paris in 1884, and afterward by Ford in New York. The posterior roots consist of four or five threads in each group. Of these, at first, all were divided after being raised on a tenaculum hook from the cord.

It has lately been proposed by Taylor to make hemisection, dividing only half or three-fifths of each group, thus modifying the sensory disturbing reflex but not destroying the pain sense and consequent poor co-ordination of the motor function. This note has been the sequel of Sherrington's observation on monkeys, that when all the posterior roots were cut the animal refused to use his hand to feed himself, though cortical stimulation showed perfect muscular function.

In my last case, to be narrated soon, I did this hemisection of each root with the happiest results. Förster has emphasized the wisdom of not cutting the posterior roots of a large number in continuity with the same idea of producing a not complete anaesthesia but sufficient to control sensory reflex. With this idea he advises, if the legs are spastic, that, instead of the lumbar 1st, 2d, 3rd, 4th, 5th and 1st sacral, one should omit the 4th; or the 1st, 3rd and 5th; or the 2nd and 4th, as seems wisest at the time.

A better suggestion, however, is Taylor's, that half, or more of each root contributing to the sciatic or the cervical plexus, be divided. This preserves the nutrition of the remnant of the root and distributes the anaesthesia evenly.

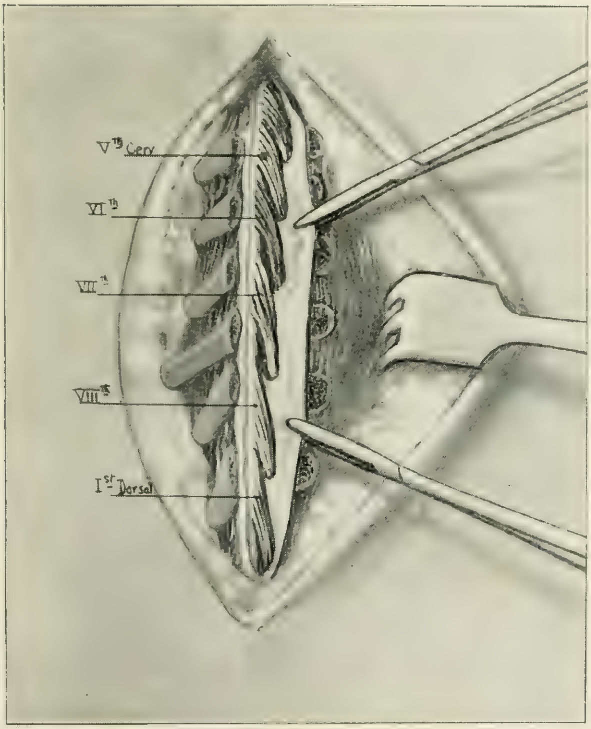
This was most efficient in the last case I am about to report and will probably be accepted as the simplest and wisest method.

The condition of anaesthesia following this operation is not uniform on account of the overlapping distribution of the root terminal filaments. There is rarely complete anaesthesia.

So many of the cases which have come to operation where good section has been made have had previous amputation of the limbs that little accurate study has been made as yet. In working out the revised methods also partial and irregular sections have given puzzling and paradoxical results. In the main, however, there has been a partial anaesthesia of the skin corresponding to the root supplying these. In some cases there has been a partial restoration of sensation.

In reporting my own five cases together, all having been done at St. Luke's Hospital, I will make the briefest mention of the three reported at length in my Boston paper, 1889.

Case I—M. L. T., Man of 40 years, suffered 2 years from violent spasms of pain in the right arm with spasmodic drawing up of the arm. This followed remotely from a shell wound of the shoulder during the war but more immediately from a long exposure to cold in an ice refrigerator in which he was renewing a zinc lining. He had had nerve stretching, nerve section and finally a low and a high amputation near the shoulder—before he came to me. He had acquired morphine habit mildly. Dr. Seguin thought the pain largely delusional with an acquired morphine habit. All other neurologists regarded it as due to ascending neuritis. The man gave evidence every five or ten minutes of sharp pain in the stump of the amputated arm, usually causing him to double over and grasp the stump with his left hand. When asked to describe the attack, says it jumps and the stump draws to his side when the pain shoots into his hand and fingers, as if they were still on, and he can feel them all drawn up. He avers that it keeps up during the night.



Incision for resection of posterior roots of spinal nerves.

Dr. Dana, Starr and others diagnosed neuritis and Dr. Dana devised the root section operation. It might be called "Rhizotomy." I divided the 6th and 7th nerves on emerging from the dura but inside the vertebral canal. Pain was not measurably relieved. Two days later I laid the patient face down under a bright light and on removing the packing from the wound, split up the dura and picked up and cut the posterior roots only of the 7th and 8th nerves. As the 6th had been included in the section of the whole nerve outside the dura, it was evident I had divided the sensory roots of the 6th, 7th and 8th, which distribute to the hand and forearm. During the operation Dr. Dana frequently applied a fine electrical stimulation to the roots before cutting, thus identifying muscular distribution. This seems not to have been done in other cases, but may wisely be used in doubtful circumstances with safety. A one-cell dry pocket battery is sufficiently strong. For ten days after operation the patient had more or less pain but of entirely different character. It no longer went down into the hand and arm, nor up on the shoulder. I believe this post operative pain can be attributed to pressure on the posterior aspect of the cord—by the soft parts where the bony arch had been wholly removed. In ten days he sat up and stopped his morphine entirely. In the third week he walked freely in the open air and had much less pain sense than when he was using morphine. Anaesthesia extended from the stump up on the deltoid and across the shoulder from the center of the clavicle to the entire scapular region. This area showed striking irritability until the eighth day. If brushed over, it quickly suffused and remained so, but if pinched it blanched and remained so two minutes. This afterward disappeared.

Subsequent History—For two years he abandoned morphine and gained twenty pounds. Four years later he complained again of some return of pain in the stump and a drawing feeling in the amputated fingers. He was now seeking an increase in pension and renewed his morphine taking. Eight years later he still avers he has drawing pain in his amputated fingers, and takes 1½ grains of morphine daily. The anaesthesia remained the same. He obtained his pension increase, and lived about the same till 1910—22 years.

Case II—G. Z., a man of 45. In 1886, exposed his arm in a car window while perspiring; in a few days had sharp pain between thumb and index finger, soon extending up the forearm and causing severe intermittent pains running into fingers. After two months he grew weak and nervously exhausted from the paroxysms of pain which he also called a "drawing pain." He acquired the habit of crying out aloud with each paroxysm. Dr. Stimson, Fluhrer and Gerster, in succession, stretched and resected his ulnar and brachial plexus with aggravation so that his attacks came every five minutes in the day and every half hour at night. His shouting with pain made him a dread nuisance to his neighbors. He grasped his arm and walked the floor in agony. Sleep was fragmentary. Required $\frac{1}{6}$ gr. morphine every two hours to be even comfortable.

I operated at St. Luke's Hospital and divided all the posterior roots of the brachial plexus, within the dura. His pain abated slowly. In four weeks he was discharged with moderate improvement in pain but taking no morphine. Two months later he had less pain, stopped crying out and used no morphine. Anaesthesia was complete below the deltoid insertion. His physician thought the amount of real suffering was now insignificant. A year later he was having a mild type of the same pain. Dr. Thacher reports of the resected nerve roots: "Inflammatory exudation quite marked at portions of the surface and less marked at a few points inside some of the roots. The changes are most marked in and around the root of the 1st dorsal."

Subsequent History—He became an inmate of a home and some years later his physician said he had occasional paroxysms which make him cry out, but unless some one is watching him he will play cards for hours at a time without sign of pain. He never requires anodynes and what he still calls pain is a memory sense. He lived 17 years. This arm was atrophied. The skin became hypersensitive, and the fingers had a waxy glazed appearance.

Autopsy showed marked arterio schlerosis. The left ulnar nerve showed a ¾ inch turnefaction the size of a hazel-nut, and a second fusiform mass $\frac{5}{8}$ inch long. The nerve was oedematous and bound by adhesions. This lesion may have been the sequel of early stretching.

Case III—M. L. H., florist, aged 40. Had infantile hemiplegia with athetoid paralysis of right arm and hand and partly of leg. Constant excessive athetoid movements of the arm and increasing neuralgia led to amputation of the forearm, stretching the brachial plexus and finally shoulder amputation by three prominent surgeons, long before I saw him, yet his pain and incessant spasm, drawing his shoulder stump high up in his neck continued. Neurologists continued hopefully to apply every form of electrical stimulation, without avail.

I was asked to stretch the cervical plexus, but instead, in June, 1894, I resected a quarter inch of the posterior roots, intradurally, of the 5th, 6th, 7th, 8th and 1st dorsal all that contribute to form the brachial plexus, and, on account of the persistent shoulder spasm—as his arm had gone—I also cut the anterior motor roots of the 7th and 8th cervical which made the stump jump.

Subsequent History—At four weeks he was discharged, with very great improvement. He still had a small amount of pain sense, but the constant athetoid spasms had gone, and the stump could be handled freely without being thrown into violent spasm. He could raise and lower his shoulder at will, which had been impossible before operation. For four years I watched him. He had a lingering memory of pain and said he suffered, but the old athetoid spasms had not returned.

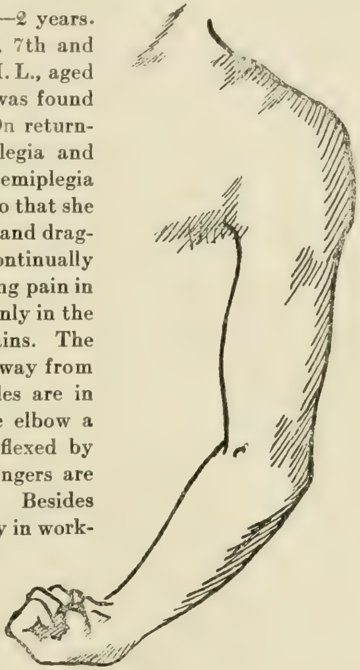
Case IV—A bookkeeper, aged 24 years. More than one year had shooting pains in his right forearm, which in six months extended to the upper arms, shoulder and scapular. Numbness and tingling of the hand and fingers followed. A tumor has been felt in the axilla six months. Examination shows atrophied muscles and paralysis of entire arm, and a large inoperable sarcoma of the axilla and sub-and supraclavicular region. Pain was unbearable and required morphine.

On April 23, 1909, I divided the posterior roots of 5th, 6th, 7th and 8th cervical and 1st dorsal with great care not to injure the anterior roots. The patient had immediate and marked relief of pain. Anaesthesia of the hand and arm up to the deltoid and axillary line was absolute. Examined by Dr. Bailey one week after finds total insensibility of the arm involving joints as well as skin. Over the deltoid sensation was blunted. In the side opposite the operated arm, there was some hyperaesthesia, and the hand opened and closed slowly. The right eye showed marked evidence of disturbance immediately after operation, which never after changed. The pupil was small and immovable, the palpebral fissure smaller and the eyeball a little sunken with a little flattening of face.

Subsequent History—The patient lived three months after operation and died of extension of the sarcoma in the neck. His pain sense was present, as it might well have been from cervical pressure, but he never lost the paralysis of motion and sensation in the arm which followed the root section. The case justifies fully the operation for relief of such unbearable pain.

The autopsy showed extension of sarcoma into all the nerves of the cervical plexus and into the very roots which I had cut.

Case V—Spastic hemiplegia of the left arm—2 years. Hemi section of the posterior roots of the 6th, 7th and 8th cervical; cure of the spasm and pain. M. M. L., aged 39. Two years ago she went to bed well and was found unconscious for three hours in the morning. On returning to consciousness had complete left hemiplegia and aphasia. The latter lasted three days. The hemiplegia continued, but gradually recovered in the leg, so that she now walks with a cane, the foot turning inward and dragging. The arm never recovered but has been continually subject to spastic flexure and constant dull aching pain in arm and hand. The pain is complained of not only in the muscles but joints. The leg also has moderate pains. The arm is paralyzed except that she can raise it away from the body at the shoulder. The pectoral muscles are in spastic contraction. The biceps also holds the elbow a little flexed, but above all the wrist is sharply flexed by the spasms of forearm flexor group, and the fingers are drawn in upon the palm—in varying degrees. Besides the disuse and pain, she is annoyed by difficulty in working a glove on the hand, which takes her about an hour. The pain alone justifies her in seeking relief. Dr. Pearce Bailey referred her to me for operation.



On June 25th, 1910, under ether, I denuded the left half lamina from the 5th cervical to the 3d dorsal and removed only one side, leaving the spines untouched. Opened the dura, picked up and divided three of the five strands composing each posterior root of the 6th and 7th and two of the four strands of the 8th root. Closed the wound. Immediately following operation the spastic contraction ceased and the fingers could be extended. During the first week I found her sitting up admiring her hand spread out on her pillow to her great delight, the fingers straight out and the wrist like the well hand—for the first time in two years. The pain also rapidly disappeared. No gain in power of the hemiplegic arm was noted.

Subsequent History—On January 2d, 1911—seven months later—she writes me: "Thank you for the relief you have given me—the pain in my hand and arm are greatly relieved; also the tension (spastic). The action is no better and the arm hangs useless at my side." Inasmuch as the paralysis had been absolute—except for the spastic contraction for two years—we can say that all has been gained that could be expected from the nerve section: relief of contraction and constant pain, especially the joint pains. The absence of complete anaesthesia due to hemi section of the roots is a decided improvement—permits all the restoration of muscle possible in a paralytic which would be vitiated by contracture of opposing ones.

Case VI—By the kindness of Dr. Lyle, I am able to include a report of a case recently operated upon by him, presenting features of interest. A young engineer was violently injured by the lever of a heavy fly-wheel striking him in the right side of his neck at the level of the jaw—in July, 1910. He was unconscious for twenty-four hours. The right arm was completely paralyzed and he suffered intense pain in the neck and arm. An exploratory incision showed that the roots of the cervical plexus were torn. The arm was then amputated. Six months later he was still suffering great pain. The right eye showed sympathetic paralysis of the eyelid with contracted and undilatable pupil.

In December, 1910, at St. Luke's Hospital, Dr. Lyle divided the 6th, 7th, 8th and 1st dorsal posterior roots within the dura, using the unilateral lamina resection to expose the dura with ease. The pain was at once completely relieved—but after three months pain has come as if in his amputated thumb. The former pain had been more or less constant before operation, like a neuritis, preventing sleep. The arm had been amputated so that no notes regarding anaesthesia are available.

Conclusions—The value of the Dana operation will depend on the proper application of the principle of interrupting the sensory reflex supplying either extremity, in cases of inveterate pain, spastic paralysis, possibly in wry neck or grave ataxia pains. The simplified technique consists of an incision an inch longer than the spinous processes of the vertebrae to be cut; separation of the muscles from one side, and unilateral laminectomy. Incision of the dura the full length of the exposure, raising the posterior roots of all the nerves supplying sensation to the part; and partial division of each root: followed by immediate suture of the dura by fine catgut: closing the external wound and drainage of the intradural space by a small tube for 48 hours. The results justify the careful and repeated trial of this method for a large consecutive series until its exact usefulness can be ascertained.

END-TO-END INTESTINAL ANASTOMOSIS BY THE INVAGINATION METHOD.*

CHARLES L. GIBSON, M.D.

Attempts to obtain end-to-end reunion of a severed intestine by invaginating one end into the other have been described since the earliest history of intestinal suture. Beginning with Jobert and Ramdohr all kinds of modifications have been practised; but it is fair to say that today intestinal reunion based on such a procedure does not occupy a definite place in the technic of intestinal surgery. The method which I venture to present has no pretension of competing with conventional methods; but it is believed that it is useful in certain difficult cases of reunion of the large intestine where other methods would ordinarily be impracticable or predestined to failure.

I first had occasion to employ it in a crude form in 1904; the report is briefly as follows:

Case I.—Nellie W., 33, colored; operated on in St. Luke's Hospital, December 19 for double pyosalpinx. The operation presented unusual difficulties, and at its termination it was seen that about six inches of the upper part of the rectum had been completely denuded of its peritoneum and its vitality obviously jeopardized. Ordinary resection being judged impracticable, the gut was divided at the lower end of the denudation. A long pedicle clamp was then passed through the anus and made to grasp the upper segment and pull it through the lower end down as far as it would go—nearly to the anus, where it was maintained by fixing the clamp in position. Two or three sutures were introduced at the point of contact of the two segments to ease the tension on the clamp. At that time the idea that one could deliberately make a satisfactory suture which could be depended on alone did not occur to me.

To my astonishment the patient made an uninterrupted recovery. The clamp was removed in two days; feces were passed normally *per anum;* there never was any leakage, as we could easily convince ourselves, the wound having been only partially closed as a precautionary measure.

In June, 1909, in the course of a panhysterectomy for fibroid uterus and a complex intraligamentous cyst, in order to make a clean enucleation I had to do deliberately what I had done inadvertently before

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*Read before the American Surgical Association, May 4, 1910.

—denude the rectum to a dangerous extent. This time I determined to make a careful and systematic reunion according to the technic to be described later.

Case II.—Mrs. O., 26; operation in St. Luke's Hospital, June 10, 1909.

The denuded rectum was divided as described in Case I. About five inches of the denuded upper end were passed down into the cavity of the lower end, and approximation of the two portions at the neck was performed by some interrupted silk sutures completed by a superimposed continuous suture. The vagina was left open and gauze packing was placed in the cul-de-sac.

The patient made a good recovery; the suture was, however, not perfectly tight as for a few days there was some fecal soiling of the packing. Discharged July 11, well healed and having natural movements. Remains under observation and continues in excellent condition, particularly as regards the bowels, an obstinate constipation existing before operation having disappeared.

Encouraged by these results I decided on this procedure as the method of choice in two resections of the rectosigmoid junction for carcinoma.

Case III.—Mrs. J., 53. Operation in St. Luke's Hospital, June 15, 1909. Extensive involvement of the sigmoid and a loop of small intestine by contact. Resection of sigmoid and twelve inches of the ileum. Reunion of the colon as described in Case II. Patient succumbed three days later. Post-mortem examination of the wound showed no peritonitis or suspicion of fecal extravasation.

Case IV.—Mrs. R., 49. Operation at St. Luke's Hospital, December 10, 1909. for intestinal obstruction of twelve days' standing. Median laparotomy disclosed a small annular carcinoma at rectosigmoid junction. Cecal anus established by a small incision in the right linea semilunaris. Twelve days later resection of the carcinoma, end-to-end anastomosis by invagination. Successful result, but some infection of the abdominal wall by a minute leakage which persisted only twenty-four hours. The cecal fistula closed spontaneously within a few days. Remains under observation, is in good health, and bowels move regularly.

During the summer of 1909 Dr. H. K. Kellogg conducted some experiments for me on dogs, which gave us also the opportunity to investigate the possibility of using this anastomosis in the small intestine, and it was found to work well; but its usefulness is perhaps more evident in the large intestine, where end-to-end union is so apt to be imperfect even in the hands of the most competent. I believe that if lateral anastomosis is impossible after resection of the colon, the method here described will be found the next best. It has also an undoubted superiority over lateral anastomosis in its simplicity and requires much less time for its performance.

Technic.—The technic here described is based on the knowledge accumulated in the cases just described and the animal experiments performed in the Loomis Laboratory of the Cornell Medical College.

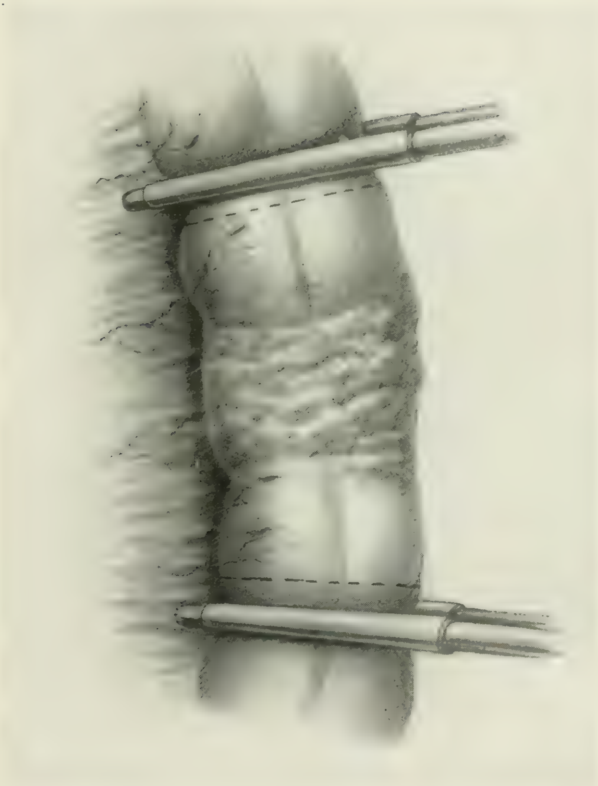


Fig. 1.—Preliminary clamping of intestine before its division.

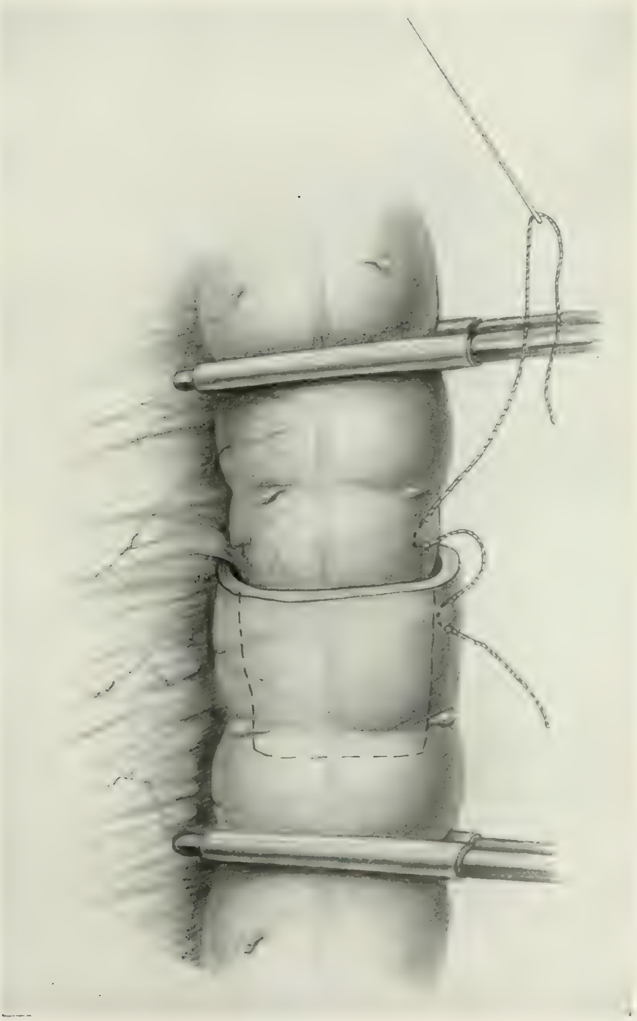


Fig. 2.—Upper segment telescoped into lower; first suture introduced.

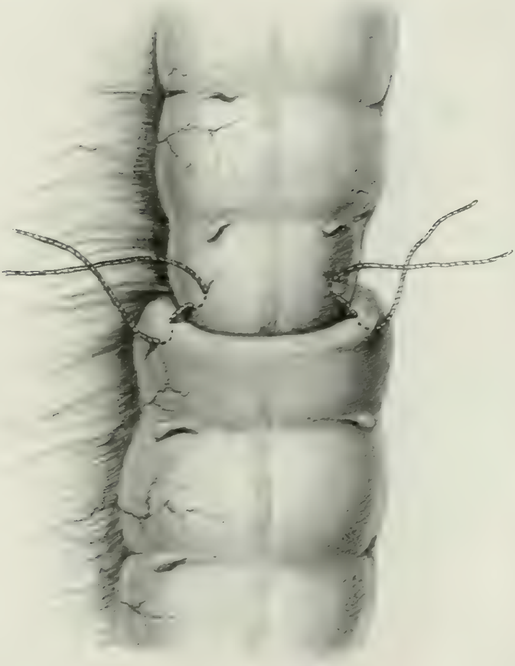


Fig. 3.—Shows invagination of edge of lower segment, bringing the two peritoneal surfaces into contact.

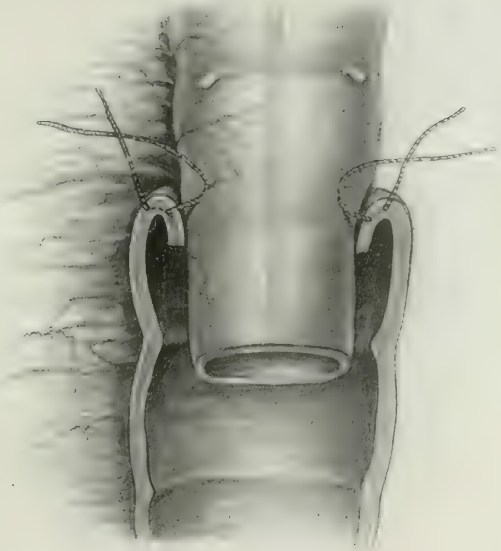


Fig. 4.—Interior view of the invagination.

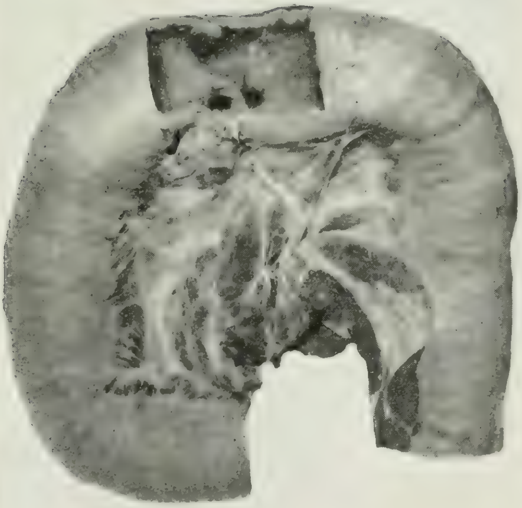


Fig. 5.—Interior view of the same specimen.

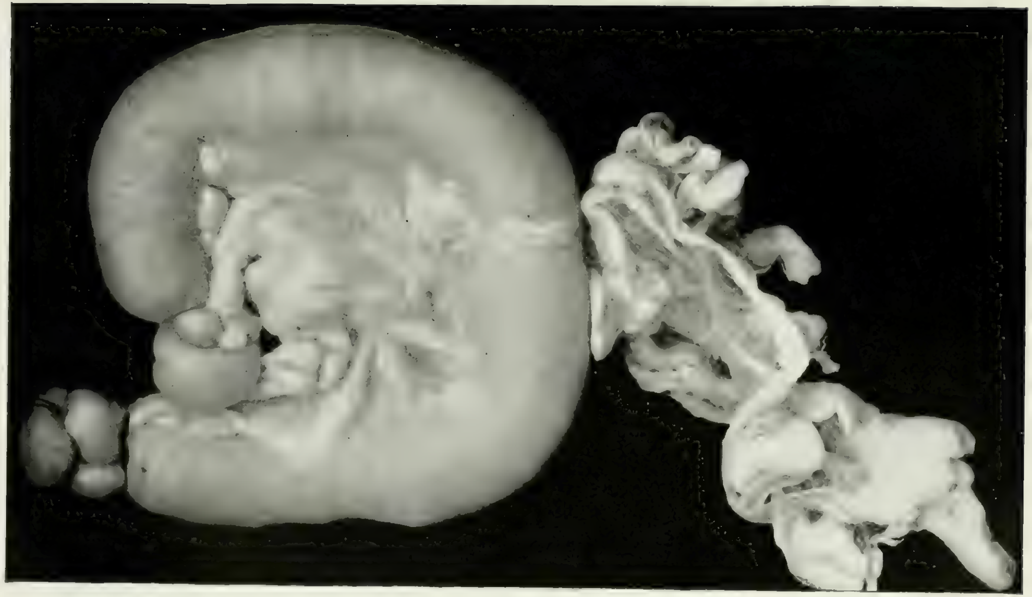


Fig. 6.—Resection of small intestine in a dog killed three weeks after operation. Shows absolutely no constriction.

Usual preparation and incision of the abdominal wall at the most suitable point. Temporary occlusion of the gut by clamps.

The upper cut edge of the gut is seized with two Kocher clamps and introduced by these into the lumen of the lower end and maintained there by an assistant. The extent to which it is feasible to accomplish this invagination will vary, depending on the laxity of the mesentery. If the latter is very short, it may be elongated somewhat by a generous incision of its outer layer. As a general rule I should wish to carry the cut end of the upper segment so far down as possible, hoping more efficiently to direct the fecal current away from the suture line. The gut is rotated about a quarter circle so that the non-peritoneal covered surfaces do not entirely approximate in the circumference. Eight to twelve interrupted silk sutures are introduced thus. A Lembert suture is begun on the lower segment, the needle issuing just short of the cut edge; on the upper segment the needle is introduced just above the line where the cut edge of the lower segment lies against the intact wall of the upper. When the knot is tied the free cut edge has been turned inward and only the peritoneal surfaces are in contact. A continuous running suture is applied over this area, further invaginating the first ones, the Kocher clamps being previously withdrawn.

In the after treatment the bowels are not moved for three days and only a low residue diet is given.

Finally, I desire to call attention to the specimen of the resection of the small intestine in a dog. There is no narrowing of the canal, and this fact dismisses any theoretical arguments that might be raised. In the large intestine such a degree of stenosis, if any, is insignificant.

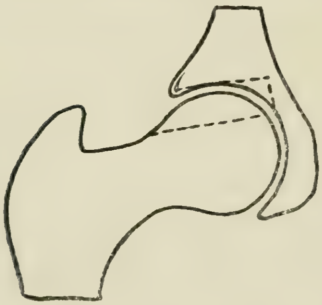
Since this paper was written, the procedure has been employed in two cases of gangrenous femoral hernia. One recovered fully, but the other died, peritonitis developing on the fifth day.

ALBEE'S OPERATION FOR ARTHRITIS DEFORMANS OF THE HIP.

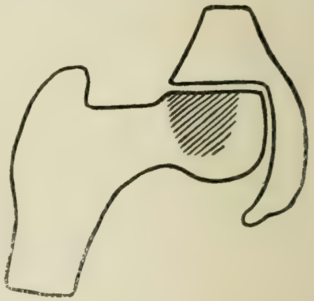
H. H. M. LYLE, M.D.

Five operations, the first performed by Dr. Albee, the remaining by Dr. Lyle. All the cases were kindly referred by Dr. Austin W. Hollis, and were treated on the Surgical Service of Division B, Dr. Chas. L. Gibson, Attending Surgeon.

The object of the operation is to produce a painless ankylosis at the hip. The operation is best preceded by an open tenotomy of the abductors as they usually offer considerable resistance and hamper the after treatment. The hip joint is exposed by an anterior incision five inches in length which begins just below the anterior superior spine, runs downward and inward parallel to the inner border of the sartorius. The sartorius is retracted outward. The inter-muscular space between the tensor fasciae femoris and the rectus is separated by blunt dissection. This space leads at once down to the neck of the bone. The surrounding ring of osteophytes is cleared away exposing the acetabular



The broken lines indicate the amount of bone to be removed. It is removed from the head and the acetabulum in different planes in order to secure the desired abduction of the thigh, when the freshened bone surfaces are brought together.



Apposition of the freshened bone surfaces after the removal of the bone from the head and the acetabulum and the femur placed in slight abduction. The blackened area indicates where the cartilage is removed when the femur is strongly rotated outward for that purpose.

rim. The over hanging portion of the acetabulum is removed, the dome being converted into a flat bony roof. With the head *in situ*, the corresponding portion is removed from the upper surface of the head, thus leaving a flat bony base. The leg is rotated outward, as much of the cartilage as possible is removed from the surface of the head and acetabulum, the bony surfaces are then brought into contact by abduction and the wound closed without drainage. See Fig. 1 and 2.

Both legs are put up in a Stiles' abduction splint. The splint extends from the axilla to the toes, the object being to maintain the leg in a position of abduction, ten degrees inflexion and moderate inward rotation.

The advantages of the operation are obvious. It is simple. There is no dislocation of the head from the acetabulum, consequently very little or no shock (a very important point in old people). It yields a firm, painless ankylosis.

The difficulties are: 1—The ring of osteophytes in the advanced cases may mislead with regards to position of the acetabulum; 2—Maintaining the corrected position.

Case No. 1.—J. K. A previously active business man, 63 years of age. Past history negative. Twelve years ago, a sidewalk over which patient was walking broke, letting his right foot through. The right hip received a "severe jar." Soon after this, he began to have pain in the knee and thigh, especially while walking, Ten years ago, he began to have pain in the region of the right sciatic nerve. This has also gradually increased in severity. Three years ago a severe pain began just posterior to the hip joint. This continued to increase until for the past three months the patient was practically confined to his chair or bed. He lost interest in things, refused to go about, and retired from business.

Physical examination. Very large man. Height 5 feet 11 inches. Weight 220 pounds. Right thigh much atrophied and adducted. Walks with difficulty, hopping along as if it were very painful. Flexion limited to about two-thirds its normal arc. Abduction and rotation nearly absent. Limb adducted. Actual shortening three-eighths of an inch, practical shortening, one and one-half inches. Joint sensitive to the extreme of motion. Patient locates most severe pain as just posterior and internal to the hip joint. He was admitted to St. Luke's Hospital, Private Pavilion. The operation was done February 20, 1908. The head had slipped up on the acetabulum and was apparently progressing toward a complete dislocation. There were many osteophytes and much disorganization of the joint.

The convalescence was uneventful. This wound, as well as all the others, healed by primary union. The patient was walking with the aid of crutches at the end of four weeks, and could place his whole weight upon the operated leg without pain. The spica was changed to a short one at the end of five weeks. It was removed at the end of eleven weeks. This patient was seen June 1st, 1908, when he stated he had not experienced a twinge of pain in his hip since the operation. His family stated that he had undergone a great change mentally since relieved of his constant pain, and that he was much more cheerful than formerly.

In a letter, January 12, 1909, the patient states: "I am very much pleased to be able to report that the operation has been a wonderful success. All pain has left me, and you may remember that I was very anxious about my right knee joint, which has pained me so many years and I feared might have become chronic, but now this knee is as strong as the other, and entirely free from pain. I can walk all day if necessary. The result from the freedom from pain allows me to attend to my business and I feel at least twenty years younger."

Case No. II.—Mrs. M. U.—Chief Complaint: Pain in left hip and knee.

Present History: For past three years has had sharp pain in left thigh and some pain in left knee. No tenderness or redness. Pain and stiffness have steadily increased until at present she is unable to bend at left hip.

Past History: Unimportant.

Family History: Negative.

Surgical Condition: Left hip—Increase in bony element around hip. Marked atrophy of left thigh and calf. Left thigh flexed, abducted and rotated in. Moderate tenderness over hip. Abduction and extension markedly limited. Marked joint-crepitus.

Skiagraph shows an obliteration of space between joint surface and many surrounding osteophytes.

Operation February 6, 1909. Patient allowed to walk on 65th day. Firm ankylosis of hip. Pain in knee improved.

Case No. III.—Mrs. A. M.—Houseworker. Chief Complaint: Pain and stiffness of left hip.

Present History: Two years ago fell striking left hip. For the first two or three weeks had very little or no trouble with it. Later it was stiff and painful in the morning but by night this condition would wear off. The pain and stiffness have steadily grown worse. At present patient cannot walk without pain. If she sits down to rest, the hip becomes so stiff that she cannot rise again. Lately the joint has become sensitive to pressure. No other joints are involved.

Past History: Has never had rheumatism.

Family History: Negative.

Surgical Condition: Left hip is in position of flexion, adduction and slight inward rotation. Pelvis tilted down on left side. Marked limitation of all the movements of joint. The skiagraph shows a typical osteo-arthritis of the joint.

Operation August 26, 1909. Albee's Operation, by Dr. Lyle.

Present Condition: Firm, painless ankylosis of hip.

Case No. IV. J. Aged 46, formerly a soldier in the English army, admitted to the hospital on Aug. 14, 1909, with the diagnosis of arthritis deformans of the right hip.

The only point of interest in his family history was that his mother had suffered from rheumatic gout. The patient had had lues and gonorrhoea when he was twenty-one years old, and enteric fever in India 20 years ago.

Sixteen years ago he first noticed pain and stiffness in the right knee and thigh. This pain, which the army surgeon called sciatica, bothered him at irregular intervals; it slowly but gradually grew constant, and the pain and stiffness seemed to settle in the right hip. After serving through the Boer campaign his condition grew worse, and he was invalided out of the service with the diagnosis of rheumatic gout of the right hip. He came to this country five years ago, and for the past two years has been unable to work, as he could not walk without severe pain, and motion was limited. Splinting, rest in bed, baking and the usual rheumatic remedies having failed, he was transferred to the surgical side for operation.

Upon examination, the patient complained of pain and stiffness in the right hip. He walked with a limp, with the thigh flexed and abducted. The right hip was more prominent than the left, the great trochanter rising above Nelaton's line. There seemed to be an increase in the bony elements around the joint, with distinct crepitus and creaking. Rotation and abduction were painful and markedly limited; hyperextension was impossible; there was moderate flexion, with some muscular atrophy. Practical shortening, one and one-quarter inches; bony shortening, half an inch. The skiagraph showed that the spaces between the articular surfaces were greatly narrowed, with a ring of osteophytes surrounding the acetabulum.

Albee's Operation, August 26th, 1909. The hip joint was exposed by an anterior incision, starting just below the anterior superior spine, and extending for five inches along the inner border of the sartorius. The presenting osteophytes were removed. A little less than one-half of the upper hemisphere was chiselled away, the surface being made parallel to the neck of femur, and a corresponding section was removed from the roof of the acetabulum. The bony surfaces were then brought in contact by abduction; the wound was closed and the hip fixed in plaster. The cast was removed on October of 1909 on account of the occurrence of an ulcer. After this a Stiles' abduction splint was applied; later the patient was permitted to go about on crutches, which were discarded on November 13, 1909.

At the present time there is a firm ankylosis at the hip, and the shortening has been reduced to three-quarters of an inch. The patient has returned to work. He walks with a slight limp, is entirely free from pain, suffers no fatigue and considers himself absolutely well. He has not lost a single day's work in over a year.

Case No. V.—P. F. Aged 54 years, carpenter. Was former patient June, 1909. Diagnosis; osteo-arthritis of the right hip. Operation advised but refused. Was again patient in September, 1910. Diagnosis the same. Rest and baking treatment with no improvement. Operation advised but refused.

Chief Complaint: Weakness in right hip and knee.

Present History: Onset two and one-half years ago. Slight stiffness and fatigue after walking. Pain is in both hip and knee. These two pains are distinct. Pain in knee sharp and stinging; comes in flashes with long intervals; in hip radiates inward toward root of penis. Exercise relieves it for a time. Is gradually and steadily getting worse. Feels perfectly well otherwise. Appetite good; bowels regular; no cough; no expectoration; no hemoptysis. Condition has grown much worse since last admission. Patient now comes of his own free will for operation. States he cannot stand the pain any longer.

Past History. Has never had a day's sickness since childhood. Has at times used liquor to excess.

Local Examination: Pelvis tilts up on right side as patient stands. Right toes touch ground; heel does not. Movement of right knee not affected. Movement of right hip limited in all directions by actual mechanical disability, the limitation in abduction being the most marked. Right leg, from anterior superior spine to inner malleolus, one-quarter inch shorter than left. Circumference of right thigh, two inches less than left. Circumference of right calf, three-quarter inch less than left.

General Examination. Emaciated man of 54; nervous; does not seem acutely ill. Heart normal.

X-Ray Examination shows the characteristic findings of an advanced osteo-arthritis of hip.

Albee's Operation, December 7, 1910. Result: Firm bony ankylosis in twenty-six days. No pain in hip. First time patient has been free from pain in over two and one-half years.

Summary—All the cases were marked examples of osteo-arthritis of the hip. The second case had an additional focus in the knee. Unfortunately this was not discovered before operation, as the pain in the knee was thought to be reflex. No skiagraph of knee was taken before the operation. After the operation the pain in the hip and back disappeared but the patient still complained of the pain in the knee. The skiagraph of the knee shows a mild osteo-arthritis. Condition at present: pain in the hip absent. Pain in the knee, improved. General condition: greatly improved.

The other cases have been marked successes.

These patients had exhausted all medical and orthopedic means of relief and from being helpless chronic invalids, a burden to themselves, and dependent on others, have become useful wage earners.

In the light of the results, this operation is a marked advance in the treatment of the monoarticular type of osteo-arthritis and deserves an extensive trial.

Thanks are due to Dr. Albee for his kind permission to reproduce his diagram of the operation.

COMBINED TUBERCULOSIS AND CARCINOMA OF THE STOMACH: PARTIAL GASTRECTOMY.

FURTHER REPORT WITH AUTOPSY FINDINGS TWO YEARS AND TWO MONTHS LATER.

H. H. M. Lyle, M.D.

A recent search of the literature fails to reveal any other operative case of combined tuberculosis of and carcinoma of stomach. This case was reported in the St. Luke's Hospital Medical and Surgical Reports, Vol. I, pp. 74-76.

The patient had remained well and had continued steadily at work for two years and two months. He was admitted to the tubercular service with the diagnosis of pulmonary tuberculosis and probably miliary tuberculosis of the peritoneum. While in the hospital he developed what was supposed to be an acute intestinal obstruction from adhesions. Under local anesthesia an exploratory laparotomy was performed. On opening the abdomen, considerable blood tinged serum escaped. The intestines were equally distended, no mechanical obstruction was found. The visceral and parietal peritoneum were studded with numerous small tubercles. The condition was paralytic, not mechanical. The distention was relieved by the formation of a left inguinal colostomy. The operation only afforded a temporary relief. The patient died two days later.

Autopsy by Dr. Zinsser, July 8, 1909. Body of a very much emaciated man of middle age. On the abdomen to the right of the mid-line there is a longitudinal incision, extending about 2 cm. below the ensiform to the umbilicus. In the left iliac region there is a fresh colostomy wound.

Thorax: On opening the thorax, viscera are found in normal position. The anterior mediastinum is obliterated by adhesions of the pericardium. The pericardial cavity is obliterated by adhesions between the peritoneum and the heart.

Heart: Changes of fatty myocarditis.

Lungs: Both lungs are adherent throughout their pleural surfaces to the thoracic walls. Both are thickly studded with tuberculous foci, which in the lower lobes adhere to the miliary type, becoming thicker and individually larger in the upper lobes. Here areas of phthisical consolidation are seen and a few small cavities are found. The bronchial nodes are enlarged and one or two are cheesy.

Abdomen: The peritoneum, both visceral and parietal, is studded with miliary tubercles which are apparently thicker in distribution in the upper quadrant.

Liver and spleen: Changes of congestion.

Pancreas: Apparently normal.

Kidneys: Changes of parenchymatous nephritis.

Suprarenals: Normal.

Stomach: At the site of the old operation there is a hard tumor, about 1 x 2 cm.

Intestines: Apart from the miliary tubercles on their serous surfaces, they show no lesion. Intestinal walls are extremely friable and much discolored, probably due to post-mortem conditions.

Bladder: Normal.

Anatomical Diagnosis: Pulmonary tuberculosis: miliary tuberculosis of peritoneum and of the intestinal coverings; carcinoma of stomach; parenchymatous nephritis, and fatty degeneration.

Microscopical Examination:

Lungs: Typical tuberculosis, both miliary and larger phthisical consolidations.

Kidneys: Parenchymatous degeneration of tubular epithelium and exudate into the tubules. Malpighian tufts but slightly involved.

Liver: Slightly congested, otherwise normal, except for a few small foci, consisting of acute abscesses, not tuberculous.

Pancreas: Normal.

Spleen: Congested.

NECROSIS OF THE HEAD OF THE PANCREAS.

H. H. M. Lyle, M.D.

Mr. S. Aged 22 years, came under observation August 25, 1909, with a previous history of indefinite attacks of pain occurring at irregular intervals. The pain was referred to the region of the stomach. These attacks had extended over a period of a year, and were attributed to indigestion.

Several hours prior to his admission to the hospital, he was seized with sharp agonizing pains in the stomach, associated with nausea, vomiting and extreme prostration. These symptoms persisted. He said he had had a series of chills with high fever. Upon admission his temperature was 104.2; pulse 130; respiration 24. A blood count showed 19,700 white blood-cells, wth 92 per cent. polymorphonuclears. The patient was ashy in color and had every appearance of suffering from collapse and a profound infection. The abdomen was distended, rigid and extremely tender, these signs being most marked in the epigastrium. There was no dulness in the flanks. No mass could be felt.

The case was regarded as one of perforating gastric ulcer. The abdomen was opened through a median incision and the stomach inspected. It showed some congestion, but was otherwise normal. Gall-bladder normal.

Behind the stomach, in the situation of the head of the pancreas, there was a tense swelling about the size of the fist. This was red, congested and covered with flakes of fibrin. There were no evidences of fat necrosis. After walling off the abdomen, the mass was opened and twelve ounces of bloody sero-purulent material evacuated. The mass was found to consist of an oedematous necrotic material, which was readily removed by the hand. The whole of the head of the pancreas was destroyed. A rubber dam drain was inserted and the abdomen closed.

The patient had a stormy convalescence, his temperature ranging between 104 and 106, and accompanied by a daily chill. During the second week the temperature gradually declined, falling to normal at the end of three weeks. Subsequently, the abdominal wound broke down, resulting in a hernia. Otherwise, the patient's further recovery was uneventful.

A microscopic examination of the material, composing the mass, showed it to be necrotic pancreatic tissue. Chemical findings (post operative); Urine reduced Fehling's Cammidge's test, negative; Gross' test for trypsin in feces shows a marked diminution of trypsin; Boldyreff regurgitation test—regurgitation possible, but a very small amount of trypsin present.

SARCOMATOUS DEGENERATION OF A PARASITIC FIBRO-MYOMA OF UTERUS. ACETONE TREATMENT.

H. H. M. LYLE, M.D.

Mrs. J. D. Aged 46 years, houseworker, was admitted to the hospital giving a history of persistent bleeding for the past four months, with considerable loss of flesh and strength. Up to four months previously she had considered herself perfectly well. The menstrual history had been normal, and the patient had borne four children, the last ten years ago.

Physical Examination: Patient of large frame. Fairly well nourished. Skin and mucous membrane anemic. Heart and lungs normal.

Vaginal perineum is intact. Cervix apparently normal but displaced to the right side.

On bi-manual examination the uterus is slightly enlarged, normal in position and fairly moveable. There is a large, hard, smooth mass the size of the fist in the left fornix. No relation to the uterus can be made out. There is a small pin point sinus in the vault of the vagina. This sinus seems to lead to the above mass. A sanguineous exudate can be expressed.

Preoperative diagnosis: 1—Possible fibro-myoma of the uterus; 2—intra-ligamentous cyst.

Operation September 17, 1910. With patient in Trendelenburg position a median incision five inches in length was made and the abdomen opened. Uterus normal. Bladder pushed up to the left by a mass lying in the base of the broad ligament between its leaves. The broad ligament was incised and the tumor inspected. A portion was removed for diagnosis. As this tumor appeared to have no connection with the uterus, cervix, bladder, etc., it was thought wise to close the abdomen and approach it from the vagina.

The laboratory returned a diagnosis of sarcoma. It was found to be a fungating sarcoma lying in the base of the broad ligament, just above the vagina and having no connection with the bladder, ureter, cervix or uterus.

It was clearly inoperable, due to its infiltration of the surrounding tissues.

The patient was then placed in the Trendelenburg position and the cavity filled with acetone. She was kept in this position for fifteen minutes, the excess of acetone was absorbed and the cavity packed with acetone gauze.

Patient made a good recovery and since leaving the hospital has been having bi-weekly treatments with acetone and so far has apparently held her own, the bleeding and discharge being controlled by acetone applications.

Later reports show that although the local condition is improved, she is beginning to suffer from a secondary progressive anemia.

Pathological Report by Prof. Francis C. Wood:

Diagnosis: Fibromyosarcoma.

Macroscopical—Tumor of left broad ligament between uterus and bladder, not connected with either. (Twenty pieces of tissue of different sizes. Two of them 2x1.5x1 in., others smaller, many 1x1x1.5 in.). Tissue appears semi-elastic, not very hard, gelatinous, and white in color for the most part. Some portions are rather yellowish.

Microscopical—Section is for the most part composed of sarcoma tissue consisting of rather large, oval and in some places spindle cells, surrounded by a small amount of fibrous tissue. In some places the cells and fibres are widely separated forming a delicate reticulum, and resembling myxomatous tissue. Some portions of the section are composed of a fibrous connective tissue containing a number of small round cells and resemble a fibromyoma of the uterus.

MESENTERIC CYST: RESECTION OF ILEUM.

H. H. M. Lyle, M.D.

Mrs. K. P. Aged 40 years, stenographer, referred by Dr. Bastedo, was admitted to the hospital Aug. 27, 1910. The family history and past history were negative. For the past year she had had several attacks of pain in the lower abdomen. These attacks of pain were accompanied by vomiting, distention and abdominal tenderness. No causal relation could be made out. The attack would last a few days and then clear up to return in from two to three weeks, the intervals of relief growing shorter. Menstruation had increased in frequency and diminished in amount. There was no leucorrhoea. Since the last attack she had had persistent tenderness in the left iliac region. The chief complaint was recorded as "Recurrent attacks of pain in the pelvis with irregular menstruation."

Physical Examination: Woman fairly well nourished; Good color. Superficial glands not enlarged. Lungs and heart normal.

Abdomen: Liver and spleen normal. Kidneys not palpable. Some tenderness in both lower quadrants, most marked in the left quadrant. No mass felt.

Vaginal: Cervix and vagina normal.

On bi-manual examination a large cystic mass is found in the left fornix. This mass appears to extend up and backwards into the pelvis. The relation between it and the uterus cannot be determined. The same mass can be felt per rectum.

Operation Oct. 1, 1910. With the patient in Trendelenburg position, a median incision 4 inches long was made. On opening the abdomen a mass of congested omentum and adherent coils were found. On separating the adhesions, a thin walled cyst about the size of the fist was found between the leaves of the mesentery in the small intestine. The left leaf of the mesentery was detached and the cyst enucleated up to the gut. At this point the cyst was found to be so adherent to the gut that five inches of the ileum had to be resected. The ends were united by an end-to-end suture, the wound was closed without drainage and the patient made an uninterrupted recovery.

Pathological Report: by Prof. Francis C. Wood.

Diagnosis: Thin walled cyst containing peritoneum, fibron and leucocytes.

Microscopical Examination: A thin walled cyst filled with a gelatinous material. The cyst is intimately adherent to the small intestine. Cyst (collapsed) is 5 x 6 c. m. The interior of the cyst is multilocular.

Microscopical Examination: A thin walled cyst of connective tissue without visible lining membrane. Outside is a very loose mesh of fibrin infiltrated with polynuclear leucocytes.

INJECTION OF BLOOD FOR AN OLD UNUNITED FRACTURE OF THE TIBIA AND FIBULA; RESULT, BONY UNION.

H. H. M. LYLE, M.D.

Mr. F. Aged 46 years, sailor, was taken to a New York hospital where he was operated on September 30, 1908. The fragments were reduced, the ends of the bones trimmed and the splinters removed. Three and one-half months later there were no signs of union. A second operation was done at which sections of both bones were removed and the ends freshened. This was also unsuccessful, and four months later there were no evidences of union. At a third operation an attempt was made to correct the position of the bones and freshen the ends. Orthopedic braces were applied, and the ends of the bones stimulated under gas. This also proved unsuccessful.

A year after the original operation the patient was admitted to St. Luke's Hospital, in the service of Dr. Charles L. Gibson. Examination showed an ununited fracture of the right tibia and fibula, and weak fibrous union between fragments of the left tibia and fibula. Operative and orthopedic measures having failed, it was decided to try Bier's method of blood injection combined with passive hyperemia. Accordingly, twenty c. c. of the patient's own blood was injected between the fragments. Four injections were given, and at the end of three weeks there was firm fibrous union. Three weeks later there was bony union and the leg was so strong that the patient could bear his weight upon it. Two months later patient returned to work. Examination at the present time shows a firm bony union in both bones of both legs.

LARGE INTRA-THORACIC CYST OF THYROID, CAUSING DYSPNOEA.

WALTON MARTIN, M.D.

A. M., a native of the United States, twenty-one years old, printer by occupation, was admitted to the service of Dr. Austin W. Hollis on September 27th, 1910. He was suffering from cough, headache and difficulty in breathing. He said that for the past four years he had been very susceptible to attacks of bronchitis, the slightest exposure to cold or wet causing an attack. For the past two years he had been short of breath on exertion, such as going upstairs or walking up hill.

About a year and a half ago, while dressing in front of a mirror, he chanced to cough and to his surprise, saw a swelling rise above the right clavicle and then disappear. Each time he coughed the tumor appeared. He was stout and in good health, weighing 170 pounds, and except for the shortness of breath and slight headache from time to time, felt well. He next noticed that his neck was increasing in size. He changed the size of collar from 14 to 14½, then to 15.

During the following year he had two severe attacks of bronchitis with dyspnoea, wheezing, a persistent cough and profuse muco-purulent expectoration. Since the last attack he had had increasing difficulty in breathing and was blue and breathless after even moderate exertion. He was told by his friends that his voice had become harsher. The swelling in his neck increased. He now found it necessary to wear a 16 collar. The protrusion above the clavicle on coughing was more pronounced. He had consulted a number of physicians and was told that he had a hernia of the trachea and that this hernia protrusion filled up from time to time with muco-purulent material from the trachea and bronchi.

The present attack for which he was admitted to the hospital was similar to the others but more severe. The cough, dyspnoea, muco-purulent expectoration and the wheezing were all present. At no time had he had difficulty in swallowing, nor had he coughed up blood. There had been no soreness or pain connected with swelling in his neck.

He had had the usual diseases of childhood and had had adenoids removed from his naso-pharynx two years ago. Otherwise he had always been in good health.

On examination of his neck a slight swelling was seen extending from the middle of the right clavicle across the middle line, filling up the episternal notch. On coughing, it grew suddenly much larger and then as suddenly receded again behind the clavicle. The top of the swelling was dome-shaped and gave the impression that it extended behind the clavicle and sternum. It did not pulsate.

The swelling seemed to move with the movements of the trachea to some extent moving up and down slightly on swallowing. The veins on the right side of the neck were engorged, the external jugular stood out as large as the little finger; the veins of the right side of the chest were also engorged. The throat appeared normal. The right pupil was slightly smaller than the left. There were numerous rales to be heard over both lungs and the respiratory murmurs were harsher than normal. The heart was normal, the pulse regular and of good force, about 140, the respirations 30 to the minute, noisy and wheezing in character. There was moderate cyanosis. The temperature was 103 4/5. The leucocyte count showed 16,900 white blood cells of which 78% were polymorphonuclears. The sputum showed the prevailing organisms to be a gram positive diplococcus resembling the pneumococcus.

During the next ten days the patient grew gradually worse, the dyspnoea became so severe that he was unable to lie down, the cyanosis and engorgement of the veins was more pronounced; his evening temperature varied between 104 and 105; the cough and expectoration were very troublesome; he seemed to be losing flesh and strength rapidly, and was in a pitiable condition.

He was then transferred to the surgical division. The following night he seemed a little less uncomfortable but soon all his old symptoms reappeared. From the position of the swelling in its relation to the trachea, it seemed probable that he was suffering from an intrathoracic goitre, which was compressing the trachea and causing the dyspnoea and it was obvious that unless he were speedily relieved he had not long to live.

Accordingly, under chloroform anaesthesia, a transverse incision was made across the neck about one inch above the clavicle dividing the skin and platysma. The sterno-hyoid and sterno-thyroid were severed and the sterno-mastoid strongly retracted. The dissection was carried upward and the superior thyroid vessels ligated. The bleeding was very profuse from the engorged veins. Finger dissection soon exposed the bluish wall of the cyst. The patient at this time was taking his anaesthetic badly and the cyanosis was alarming. The cyst was punctured allowing about half a pint of thin watery fluid to escape. The dyspnoea was immediately relieved. A Kocher clamp was then placed on the opening in the cyst wall and an attempt made to continue the enucleation. It soon became apparent that this was impossible as the cyst extended far down behind the sternum and sternal end of the clavicle into the thorax. The upper free portion of the cyst wall was therefore removed, allowing again about half a pint of watery fluid to escape and a soft drainage tube and a wick of gauze inserted into the intrathoracic portion. The muscle and skin were then sutured, except for a small aperture for the passage of the tube. The cyst wall was extremely thin. The duration of the operation was thirty minutes. The patient returned to the ward in good condition, the cyanosis had disappeared and the respiration was no longer labored.

For several days the bronchitis continued, the temperature on the evening of the fourth day was 104; on the seventh day, however, it was 99 4/5 in the evening and from that time on he continued to improve. He weighed at this time 115 lbs., having lost during his illness 52 lbs.

During this week the dressing was saturated with thin serous discharge. The drainage tube was removed on the fifteenth day, the discharge having become much less, and he left the hospital on the twenty-second day with a small discharging sinus in the neck. He had gained in the three weeks 13 lbs., the temperature was normal, the pulse 108, respiration 24. During the next two months he was seen twice a week, and the sinus cauterized with 95% carbolic acid from time to time, a long probe being used as an applicator. It passed behind the sternum for several inches. At the end of this time the sinus closed and he returned to his work. He now weighs 150 lbs., and is in good health.

ILEOCOLIC INTUSSUSCEPTION WITH RESECTION OF TWENTY-TWO INCHES OF GANGRENOUS GUT, RECOVERY.

W. Scott Schley, M.D.

Especially noteworthy among the many cases of intestinal obstruction that have occurred in the last two years upon the First Surgical Division, are; two cases of impacted gall stones forming absolute obstruction, both in elderly subjects, one of band occluding the lower part of the ileum in a woman 7 months pregnant, one of ileum herniated through a mesenteric opening, and one of intussusception with resection of gangrenous gut.

All of these were late operations (only one before the 6th day) emergencies thrust upon us and undertaken in desperate conditions due to delay in seeking surgical aid. All have recovered and in view of the great mortality in such conditions with delayed operation the gratification has been very great. The last of these cases, that of the intussusception, was operated the 6th day after the onset of acute symptoms and has not heretofore been reported. The gravity of this latter condition is well shown in Gibson's statistics of 1000 cases of intestinal obstruction in which he found 81% mortality with resection in intussusception and remarks, "only one case of resection of gangrenous gut was saved." The only other case I am aware of that recovered was that of E. W. Peterson of this city, operated in 1904, and making but three recorded recoveries from this condition.

Surgical No. 79,652. This patient, a small Italian child of six years was admitted December 7, 1909. She had been taken sick one week before with abdominal pain and vomiting. From the onset o pain she had vomited frequently and irrespective of attempts to take nourishment. Bowels said to have moved well day after beginning of attack and two days before entrance. No blood or unusual conditions were noted. She complains of pain all over the abdomen.

Physical examination shows a poorly nourished child, pale and with coated tongue. Chest, negative. Pulse, irregular and poor force.

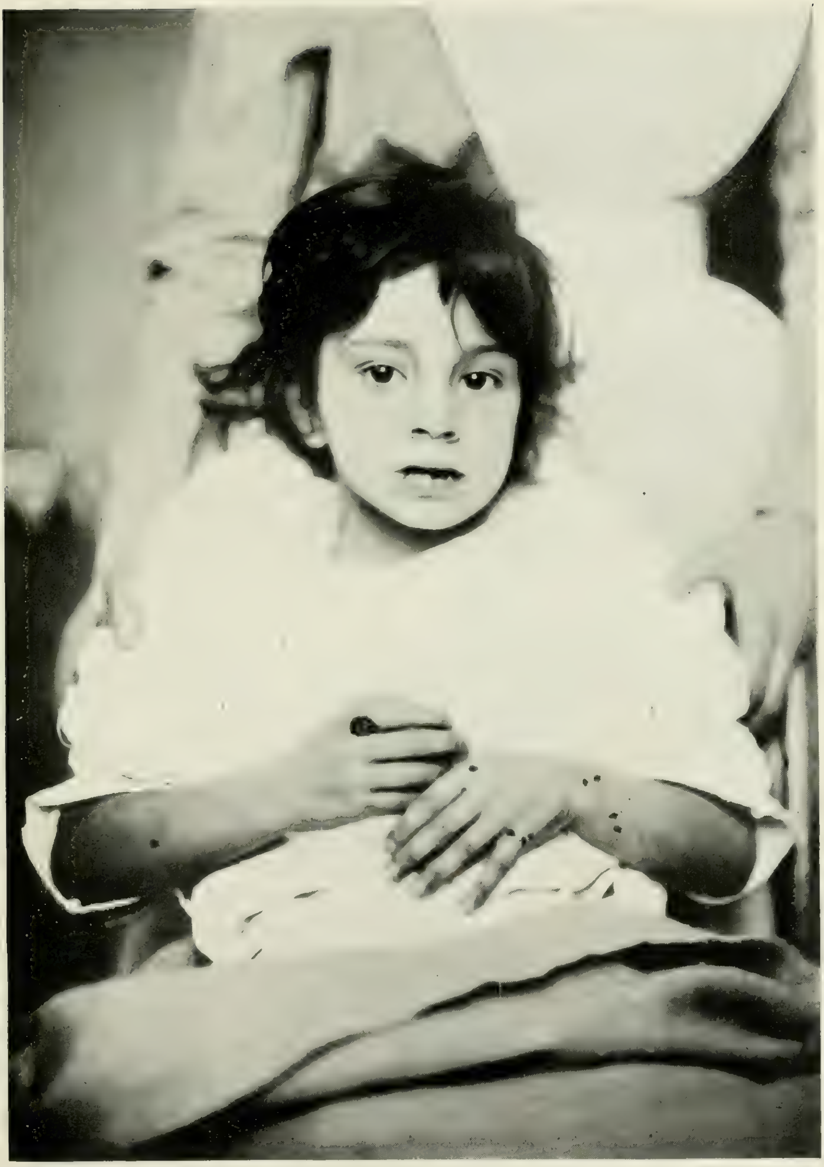


Fig. 1.—Cutaneous lesions of hands and forearms. Photograph taken shortly after operation and before emaciation had become marked.

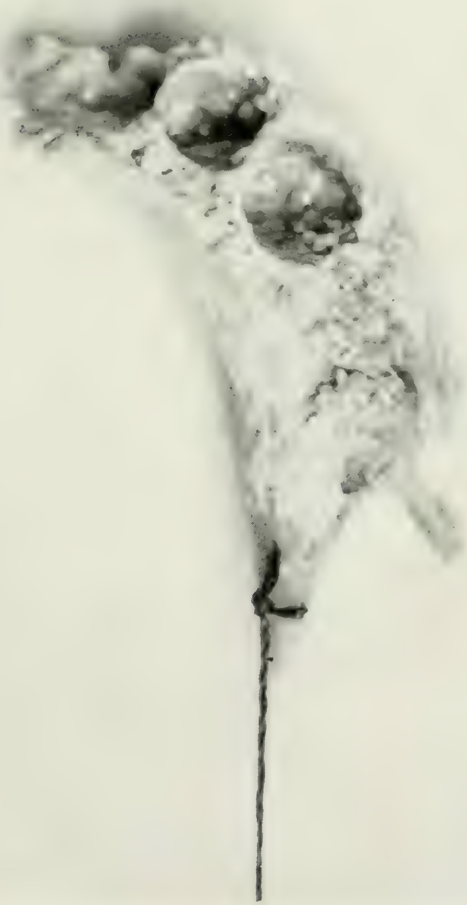


Fig. 2.—Part of necrosed alveolar border of jaw, showing sockets of two of the teeth. Photo almost twice size of specimen.

Abdomen, not rigid but moderately distended and with general tenderness. Signs of fluid within the peritoneum. Right rectus more rigid than left. Over the right lower quadrant more marked tenderness and increased rigidity. A mass occupied the region of the caput and extended for four or five inches along the line of the ascending colon. Rectal examination revealed nothing. Temperature 101, Pulse 120. Leucocyte count 26,700 with 74% polynuclears.

Operation (Dr. Schley). An intermuscular incision over the caput coli with extension by opening the rectus sheath gave very perfect access through the thin abdominal wall. A large amount of foul brownish fluid escaped on opening the peritoneum which showed marked congestion everywhere. An intussusception of the ileocolic type was found and an inch and a half of black gangrenous ileum extended back from the constriction ring. There was a distinct sharp line of demarcation between this and the healthy gut. With great care, so rotten was the gut, using traction and back pressure through the walls of the colon, a large amount of completely gangrenous ileum was reduced. The vessels of its mesentery were hard and thrombosed and the connective tissue dense and infiltrated. Twenty-two inches of gut were excised, the gangrenous process extending up to the cecum the walls of which were oedematous and thickened. The ileum was joined to the cecum at the normal opening by button the union being reinforced by a double row of peritoneal stitches. The wound was closed with small drainage tube extending down to pelvis.

From the third day following operation for fully two weeks frequent loose, foul involuntary stools occurred. Upon the third day also there appeared numerous petechial areas the size of a dime upon the hands, feet, arms and legs. Many assumed a gangrenous character and became ulcers slow in healing. The Friedlander bacillus was present but vaccines prepared from this were without benefit. A few days after these areas were noted on the skin a black necrotic spot appeared on the mucosa of the mouth under the tongue on one side and rapidly spread until it involved the anterior two-thirds of the floor of the mouth on the left side, the under side of the tongue of the same side and the alveolar border of the jaw from the second molar to the incisors. Full strength tincture of iodine was thoroughly applied for three successive days and apparently controlled the extension. The necrotic tissues sloughed, taking with them the bony alveolar process of the jaw and the teeth. The large area subsequently cicatrized with but little deformity. The Murphy button was passed on the 7th day.

The child's condition continued profoundly septic with foul stools and high pulse and temperature. Rectal and colonic irrigations were given and although the weather was severe with rain and snow she was put out of doors well wrapped up and protected. Emaciation had been progressive but after the inauguration of the out-of-door treatment she seemed to hold her own but without perceptible gain. As a further resource it was decided to transfuse her using the father as donor. On January 4th, four weeks after operation, this was done. It was difficult to find a vein of sufficient caliber, so shrunken were all her tissues, but finally the external jugular of the neck was anastomosed with the radial artery of the father, under local anesthesia. Most of the instruments devised for the purpose were of little value so that the artery was simply coated with alboline and held within the vein by forceps. Blood was allowed to flow for 20 minutes. The patient was benefited and slow gain was noticeable from that time. Irregular and high elevations of temperature continuing for a week at a time would still occur and associated with a high leucocyte count and occasionally with a high differential count, but nothing focal could ever be detected and blood cultures at this time were reported as negative. Upon December 30th the leucocyte count showed a total of 49,000 with a polynuclear percentage of 82. At this time the temperature was running to 104 F. with remissions to 100 2-5 in the mornings. For over two months however it held very steadily up and without the more marked fluctuations of the usual septic case. There were no chills at any time. The urinary findings were negative except for a trace of albumen throughout.

This child left the hospital 7 months after her operation in excellent condition. Her gain was exceedingly slow and scarcely to be noticed from week to week. The septic thrombosis of the mesenteric vessels played a large part in the condition following operation. As much of this mesentery as possible was excised at operation to get rid of this focus of infection. The peritonitis subsided during the first week but the wound remained sluggish and open for many more, making intestinal protrusion very difficult to prevent, there being no adhesion and effort at repair.

The feedings during much of the difficult period of malassimilation were kindly supervised by Dr. Gould. Cocoa butter inunctions were given daily and no effort spared to increase nutrition.

The persistent open air treatment throughout was probably the largest single factor for good during convalescence and without this

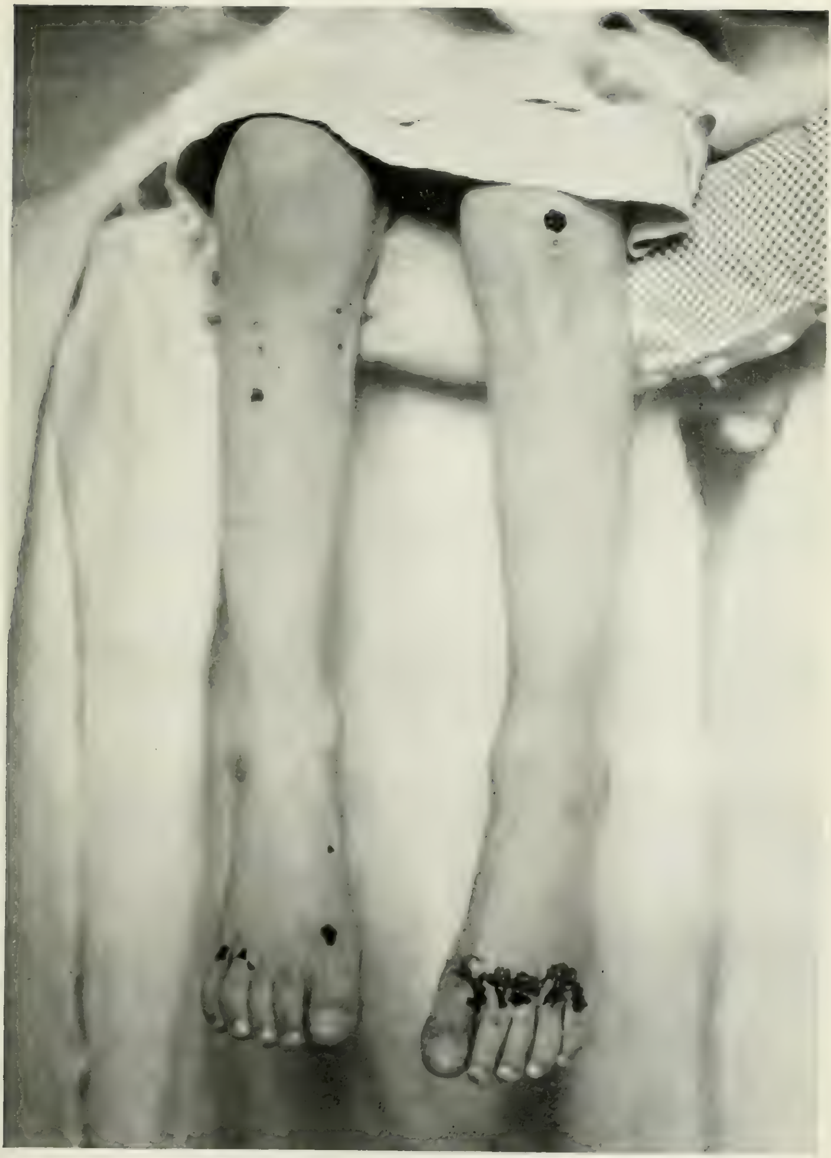


Fig. 3.—Cutaneous lesions of feet and legs.

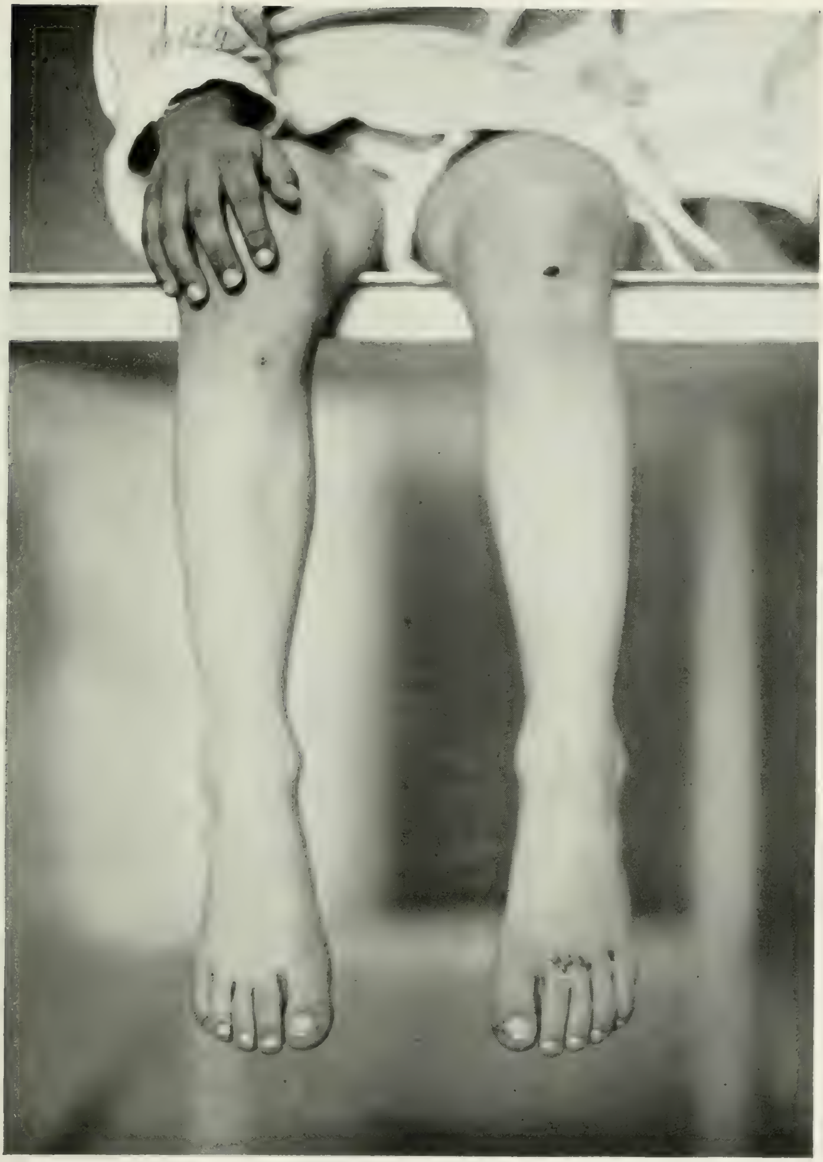


Fig. 4.—Appearance seven months later; practically healed, a few dry scabs.

and the unremitting care in the nursing a successful outcome would hardly have been possible.

The operative work occupied twenty minutes and was productive of but very slight additional depression leaving her in the best obtainable condition to battle with the toxemia.

BRASS SCREW LODGED IN RIGHT BRONCHUS, LOW TRACHEOTOMY, REMOVAL

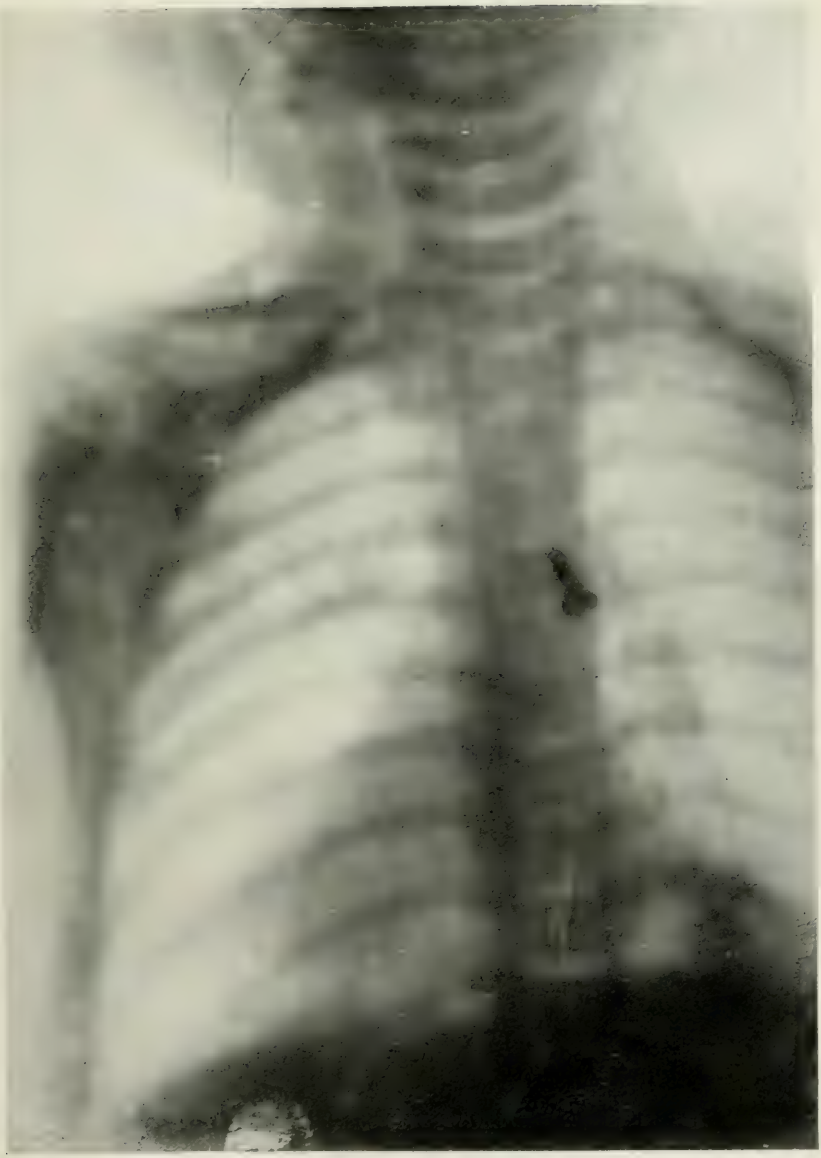
W. Scott Schley, M.D.

Surgical No. 67,705. L. F., a small Italian child of 18 months was brought to the Dispensary by the parents with the history of having "swallowed" a screw three days before. Since the accident the child has coughed much, cried continually and refused to eat. Fearing something wrong they brought her for advice.

The physical examination of the chest and a fluoroscopic inspection were suggestive of a foreign body low down in the trachea and the patient was sent up to the ward, entering the First Surgical Division. It was my duty to see this case, secure an immediate X-ray plate and operate if the diagnosis were confirmed. The breathing was suppressed and there were sibilant and a few sonorous rales over both sides and along the sternum in front. Percussion note everywhere unchanged.

The X-ray having established the diagnosis without doubt and located the screw with practical certainty in the first inch of the right bronchus the child was given a light chloroform anesthesia and a low tracheotomy done. By introducing a moderately long alligator jaw urethral forceps down the trachea and pushing gently over into the right bronchus the sharp metallic click of the instrument and screw could be plainly heard. After a few efforts it was grasped and withdrawn for nearly two inches when it slipped from the forceps and dropped into the trachea. Another forceps of the same type easily removed it from the trachea at the next trial. A tracheotomy tube was worn for two days and the wound then allowed to close under moist boracic gauze. Convalescence was entirely uneventful.

This patient was deliberately put in a half Trendelenburg position during anesthesia as well as operation to prevent any respiratory or operative act, which might dislodge the heavy piece of metal, causing



Brass Screw in Right Bronchus. Not only can the diagnosis of screw be reasonably made, but the screw is round-headed as well.

its gravitation deeper into the lung. In the inverted posture, the tendency will be greater for heavier foreign bodies to fall back into the trachea where they can be more easily dealt with.

The diagnosis in this case was confirmed by the X-ray, the operation done and screw extracted within an hour of the patient's admission to the Hospital. The skiagraph of the chest is shown herewith.

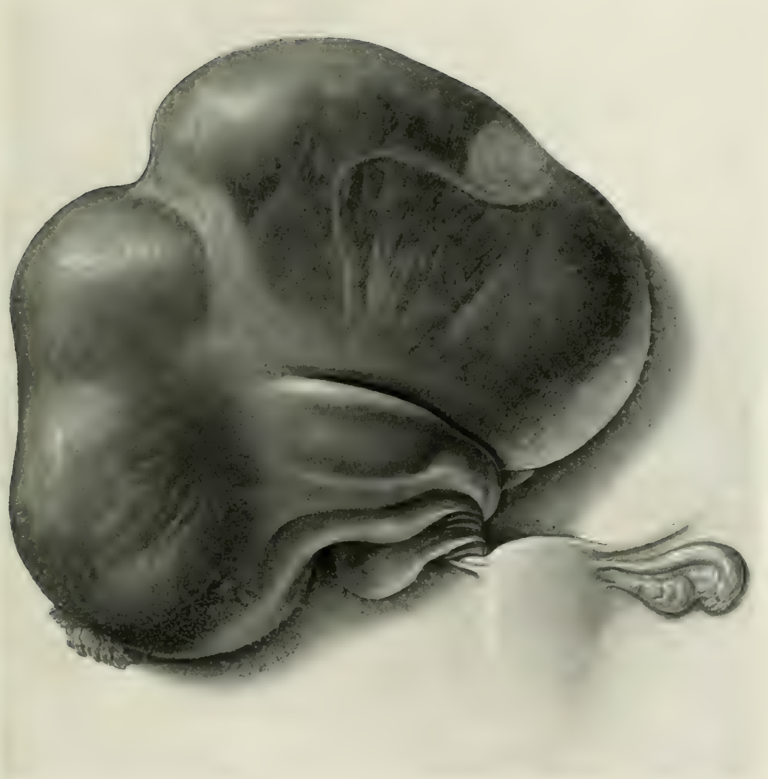
GANGRENOUS OVARIAN CYST, DUE TO TWISTED PEDICLE.

W. Scott Schley, M.D.

Case No. 84,000—M. P., a woman of 29 years of age, was admitted to the First Surgical Division, January 2d, 1911. On December 30th, three days before admission, she had been taken with pain in the left side of the abdomen which resembled that of her menstrual periods. It remained of the same intensity for 24 hours and then increased considerably in severity, involving the whole lower abdomen, which became very tender to touch. Vomiting ensued and lasted for some hours. The tumor, which she was conscious of having had for four years, seemed to grow larger. An ice bag was applied and morphine given for two days before entrance with consequent diminution of pain.

On entrance the temperature was 102 1-5, pulse 112, blood count reported as 21,300 with polynuclear percentage of 95. The abdomen was large, somewhat distended and with general tenderness. Rigidity general and moderate. A large mass, but slightly movable, occupied the lower part of the abdomen and extended two fingers above umbilicus. It was tense and elastic and not attached to abdominal wall. The uterus was behind and below the mass in the sacral hollow. The diagnosis was reasonably certain of ovarian tumor with twisted pedicle.

In incision from just above the umbilicus to the pubes revealed a large perfectly black mass, the size of a seven months' pregnant uterus. The pedicle was found with two complete turns and just below the last showed a sharp clear line of demarcation between the strangulated and healthy tissue. The tumor was dense, heavy and with much engorged surface vessels, showing a venous closure of the circulation antedating somewhat the arterial. The fluid in the abdomen was moderate in amount and consisted of cloudy serum. The patient's convalescence was uneventful and she left the hospital on the 15th day following operation. The condition is well shown in the plate made from a drawing of the specimen.



Gangrenous unilocular ovarian cyst of left side. Pedicle consists of tube and ovarian ligament, untwisted; pedicle measures an inch and a half broad by two inches in length. The tubal arrangement and peritoneal reflections simulate intraligamentary cyst.

TUBERCULOSIS OF THE BREAST.*

W. Scott Schley, M.D.

The following case of tuberculosis of the breast entered the First Surgical Division at St. Luke's Hospital in the service of Dr. Abbe.

Operation (Dr. Schley). The patient was a woman, thirty-two years of age, of English nativity, whose mother died of old age, and whose father died at seventy-six. A sister had died of carcinoma of the uterus. The patient had always been well and strong, had had five children, and nursed them without trouble with the breasts.

Present trouble was first noticed eight weeks before admission to hospital, when there occurred a slight dull pain in the right breast. Three weeks later a small lump, the size of a marble, was discovered in the upper and outer quadrant. This remained stationary in size for three weeks. Two weeks before admission the tumor began to grow rapidly in all directions, and the pain became more severe. There is no history of traumatism. Her appetite is good and she is otherwise well.

Physical examination shows an unusually well-nourished woman with good color of skin and mucous membranes. She has no cough and the chest examination is negative. In the right breast, occupying the outer hemisphere and chiefly the upper and outer quadrant, there is a mass the size of a mandarin orange, hard, coarsely nodular in feel, as though consisting of numerous enlarged, hardened, and matted glandular elements, resembling a bunch of grapes with the individual fruit packed closely together. It is not tender. It seems to occupy the site of the glandular apparatus, and to be quite movable upon the deeper structures. The skin is not adherent, but connective-tissue prolongations from it run into the mass, serving to anchor it to some extent. The breast appears of the same size as its fellow, but is slightly fuller in outline in the outer hemisphere, and there is a slight darkening of the skin over the upper half of the growth. The mass as a whole can be lifted up by the finger inserted under its edge, and when pressed against the skin the individual nodules are very apparent to the eye and blanch the overlying skin. The other breast, both axillae, and the supraclavicular glands are negative, as are the superficial glands of the body generally. The breast was amputated and the axilla cleaned out. The report of the Pathological Division is as follows:

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*Previously read before the Society of the Alumni of St. Luke's Hospital, and published.

"Macroscopical examination of the rather large breast shows that it is filled with a soft mass, some six centimetres thick, with irregular edges and numerous connective-tissue strands radiating from it throughout the remainder of the breast tissue. The central portion of this mass is quite firm in structure, and on section shows only dense fibrous tissue with a few ducts, from which a purulent fluid can be pressed. The growth resembles very closely a scirrhous carcinoma. The axillary lymph nodes are slightly swollen, but do not seem to be involved by the process.

"Microscopically, there is a moderate amount of simple adenoma of the breast. There is also abundant infiltration of the connective tissue with small round cells, and in places distinct tubercles with giant cells and central necrosis. The whole of the tumor is diffusely infiltrated with these evidences of tuberculosis, but there are no large cheesy masses and no other evidences of tuberculosis than those given above. The tubercle bacilli were demonstrated in stained sections of the tumor. The axillary lymph nodes are perfectly normal. F. C. Wood."

Two weeks after the operation the patient was given an injection of seven milligrammes of tuberculin, but showed no reaction. As a control to this test, three other known cases of surgical tuberculosis were given each six milligrammes from the same dilution and gave typical reactions.

That the case is a primary one, as far as we can at present determine during life and without a most complete and searching autopsy, I think is shown by the findings at operation, the result of the controlled tuberculin test, and (especially) by the present condition of the patient and her constant good health since her discharge from the hospital, now nearly four years ago, and living as she has under the same conditions as before her operation.

That tuberculosis of the breast is an uncommon disease, certainly as compared with the malignant and benign tumors of the same region, a search of the literature upon the subject readily shows. Of all the neoplasms of breast reported scarcely more than 100 up to 1903 were reported as tubercular. In 1904 and 1905 a number of additional cases were reported and up to 1910 about 20 more. Many earlier cases were doubtful and possible examples of simple mastitis in tubercular subjects. If we reject the cases not verified by histological examination or the finding of tubercle bacilli, the number is materially reduced to about sixty-five, in the first series of 100 cases. In an examination of these again, the number of cases in which the disease may fairly be regarded as a primary one in the mammary gland itself is further very greatly reduced to about twelve, excluding, as we must, all determinable foci of tubercular disease elsewhere,—involvement of axillary and of supraclavicular glands, visceral infection, bone lesions, etc. These occurred in the large majority of the reported cases in addition to the disease in the breast, and must throw doubt upon that organ as the seat of primary infection in those cases. There were many recorded in which the axillary glands alone and of the same side were affected; and it is in this class of case that the most difficulty is experienced in determining the starting-point of the disease. A small focus of disease in the breast, where involvement is usually slower and so small as to be overlooked, may cause enlarged and suppurating secondary glands in the axilla which may be taken for the primary or the sole lesion. On the other hand, we believe that the breast may become infected by way of the axillary lymphatics, a point that is made much of by Halstead and Le Count, Powers, Verneuil, in the collected cases of Berchold, and by Salomoni. Furthermore, the lymphatics from the pleura are believed to play a part as carriers of the infection. It is practically impossible at present to determine with certainty in such cases the original focus of disease. In considerable disease of the breast with slight or absent axillary involvement, it would seem that we may be reasonably certain of that organ as the primary source of infection. In this connection, two cases, seen in private and hospital work, are of interest. There was, in each, extensive and characteristic tubercular disease of the axillary glands of one side and of the glands along the edge of the pectoral muscle, extending as a mass up to the very margin of the breast tissue, but with no involvement of it as far as could be determined before and during the operation. None had appeared several months later. One of these patients was 53 years of age.

To the valuable papers of Halstead and Le Count and Sabrazes and Binaud I am indebted for much from the historical side of this subject.

The history of mammary tuberculosis dates from the publication of Sir Astley Cooper's[1] famous work upon the diseases of the breast, in 1829. Since that time English authors seem to have almost ignored the condition. During the following years and up to as late as 1880, a number of cases were reported but not studied histologically, with the exception of Lancereaux's[2] case in 1860, the diagnosis being based on macroscopic findings only. Nélation[3] in 1839, Berard[4] in 1842, Johannet[5] in 1853, reported cases. Velpeau[6] in 1854 distinguished three forms of the disease,—disseminated tuberculosis, lymphatic tumors, and lymphatic degeneration. Heyfelder[7] in 1851 reported the first male case (man of twenty-six years). Horteloup[8] in 1872, Poirier[9] in 1883, Demme[10] in 1889, Hebb[11] and Schede[12] in 1893, Ferguson,[13] 1898, and Delbet, quoted by Duplay and Reclues,[14] in 1892 reported cases in males. In 1860 Lancereaux reported a case before the Anatomical Society of Paris, the diagnosis being confirmed by microscopic examination. This appears to be the first recorded in which the diagnosis was so confirmed.

The second epoch in the study of mammary gland tuberculosis began with the presentation of Dubar's[15] Thesis at Paris in 1881, when the diagnosis was based not only upon histological examination but upon the finding of the tubercle bacillus. Dubar reported two new cases and described two forms of the disease,—the isolated or disseminated nodular variety and the confluent. In the first form he regarded the disease as primary in the mammary gland in the majority of cases, and in the latter as frequently secondary to disease in some other part, usually the axillary glands, but it might result from the coalescence of the nodules of the first form. Ohnacker[16] in 1883 reported two cases, the first in which inoculations into animals (rabbits) were made. Many additional cases, some completely worked out, were reported up to 1891, the year of Roux's[17] inaugural dissertation at Geneva.

Roux gave a complete review of the cases, thirty-one in number, that had been reported up to that time, and added three new ones, in two of which the tubercle bacillus was demonstrated. He also described a third form of the disease, the intraglandular cold abscess. Robinson[18] in 1892 considered the mode of origin of the disease, and concluded that it is not primarily an infection of the gland proper, but first of the connective tissue and later of the gland epithelium, the evidence being in favor of a lymphatic or hematogenous origin rather than a duct infection. Dubrueil[19] previously in 1888 had reported a typical example of this pericanalicular fibrosis with pressure and atrophy of the glandular structures.

In 1893,[20] Remy and Noel published a case of the disease in a patient of fifty-three years, the most advanced age then recorded. Powers[21] in 1894 reported thirty-five collected cases showing the disease evenly distributed throughout the third, fourth, and fifth decades of life. He found twenty-two cases in married and five cases in single women. Twenty-one of the twenty-two married women had borne children, and nine had had inflammatory troubles of the breast, six of a suppurative character. He believes that the puerperal state and subsequent lactation are not without predisposing influence upon the cause of the disease. Bull in 1894 had had but one case in 185 mammary amputations. In the 14 years following this he did not have another. Gautier[22] in his thesis in 1895 collected and analyzed seventy-seven cases. In but forty-three of these was the lesion demonstrated to be tubercular by histological or bacteriological examination. The bacillus was found in but twenty-two. Sabrazes and Binaud[23] in 1896, together with a review of the literature, presented the most complete work from the pathological and histological side that is to be found. Scudder[24] in 1898 collected eighty cases, twenty-three of which he rejects as lacking in positive evidence. One-half had borne children and the breast had been functionally active. Ten had had inflammatory troubles in the gland at some time preceding the appearance of the tubercular disease. Lactation was active during the development of the tuberculosis but a few times. Among these latter cases I have been able to find one each of Davis,[25] Ohnacker,[26] Dubar,[27] and Pisani.[28]. Halstead and Le Count[29] in 1898 reviewed the history of the subject with an excellent presentation of the symptomatology and clinical course of the disease. They report a case well worked out. Ferguson,[30] in the same year, reported the last male case. Freiberg,[31] also in 1898, and Smith[32] in 1902 reported cases that seem examples of secondary involvement of the breast by extension from the axilla through the lymphatics. In Smith's, a lump, first noticed under the arm, gradually increased in size and travelled towards the breast, which later became enlarged and discharged seropus spontaneously. Salomoni[33] and Schifoni[34] in 1901 review the literature and report cases. Bartach[76] in this year could find but sixty-five undisputed cases in the literature.

Tuberculosis of the mammary gland, then, may occur in the male or female and at any age, but is usually found in the third, fourth, and fifth decades of life and in the female, only eight male cases having been reported (three proven). It is more frequently seen after gestation and lactation. The majority of the patients were under 35 years of age, a number were under 20,—the oldest 73 (Shield),[75] the youngest under 1 year (Demme).[35] Hence it is prone to occur in young adults during the period of functional activity of the gland. In the primary cases the age of the patient and the apparent good health, frequently remarked, are noteworthy. In a case of Souplet's[36] and in one of Habermaas's [37] this was especially remarked. The relative frequency of the disease cannot be estimated from present data. In general it may be said to be of infrequent occurrence. Heredity would seem to play no greater part here than in tuberculosis elsewhere, and previous lactation and inflammatory trouble to act as predisposing causes to no greater extent than do places of diminished resistance in other parts. Injuries to the breast prior to the development of the disease have been mentioned in a few cases (Poirier,[38] Hebb,[39] Sabrazes, and Binaud)[40].

The infection must occur (1) through the ducts of the glands (Verneuil,[41] Verchere[42]); (2) through a surface wound (Demme,[43] Orthmann,[44] and Kramer[45]); (3) through the blood or lymphatic channels, the generally accepted method; or (4) through contiguity of structure, extension from caries of the ribs or sternum (Heyfelder's[46] case).

The disease process, as a rule, is slow except during lactation, affects but one side, rarely both, and the axillary glands are enlarged from tuberculosis or simple hyperplastic inflammation. More rarely they are normal. It may in the beginning present no recognizable symptoms, the gland usually preserving its normal size and contour. But it may even be small and insignificant in an advanced case, as in one of Piskacek's.[47] In comparatively few cases is the volume appreciably augmented. The skin is normal, non-adherent, and without fistulae. Nipple retraction was noted in a moderate number of cases only. Pain is not a constant symptom, and was spoken of as severe in but few of the cases. It seems dependent upon the rapidity of growth of the tumor. Direct nerve involvement has been described by Dubar[48] and Salomoni,[49] and by the former as the cause of the pains met with. In the end nearly all become painful, the breast enlarges, the growth undergoes the degenerative changes characteristic of tubercular tissue, abscesses develop and fistulae form.

In the first, the nodular or discrete form, characterized by its chronic course and painless insidious development, the nodules may be single or multiple. The breast is nearly always unchanged in appearance. The nodules are firm, movable, and distinct, or their outline may be indefinite, merging with the normal gland tissue. They may resemble lymphatic glands situated on the margin or scattered throughout the breast."*

These nodules may remain of the same size for years and then advance, or they may steadily increase in size over a long period of time. In Scudder's[50] case five years, in Mandry's[51] four years, and in a number between one and two years. If single, they are more frequently found in the upper and outer quadrant. If multiple, they may coalesce and form tumors of considerable size before degeneration and suppuration occur (as in the case herein recorded), and may become attached to the skin or pectoral fascia. Or they may remain distinct and undergo the usual changes, the breast being filled with multiple cavities of cold abscesses. Sometimes a single cavity results. The foci open externally, as a rule, after a time.

Sections through the breast, before degeneration, show distinct, firm, slightly yellowish or wax-colored nodules, from the size of a pinhead to that of a marble, and with a peripheral zone of grayish or bluish tinged, slightly translucent tissue. Between the foci healthy gland substance is seen. Microscopically, the nodules begin as an infiltration, with embryonal cells around the glandular acini and ducts which later become invaded. The centre of the mass is destroyed while the process goes on and spreads at the periphery.

In the second or confluent form, more frequently met with, there is a more acute onset and greater enlargement of the breast. Degenerative changes occur and fistulae result early, usually in less than a year, and especially in those near the time of lactation or with the tumor situated near the nipple, when it may be a question rather of weeks. Such instances occur in the cases of Habermaas,[52] Hebb,[53] Mandry,[54] Piskacek,[55] Davis,[56] and others. Bloodgood[77] in 1908 remarks; "rarely do we see tuberculosis before abscess and sinus formation."

A single tumor is usually found and situated in the outer hemisphere of the enlarged mammary gland. It presents itself as an irregular, nodular mass, varying in size from that of a walnut to that of an apple or larger, at first hard, but softening later and subsequently fluctuating. The breast may be double the size of the sound one and fistulae are frequently seen.

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*In a very early case, in the person of a hospital nurse, whom I had the opportunity to see, the axillary glands were markedly tubercular, while the breast presented but a very small, ill-defined area of induration nearly under the nipple and deep in its mass. The feeling was much that of the sense of caking found in a single lobule of the lactating gland, but with less distinctness.

In this form of the tubercular process, masses extending from the breast towards and as far as the axilla were frequently described, or the breast tumor may be joined to an axillary mass of glands by a band of indurated tissue. This was referred to in the cases of Hebb,[57] Pisani,[58] Gaudier and Peraire,[59] Salomoni,[60] Berchold,[61] and others. It is regarded as a "characteristic sign of mammary tuberculosis."

In the vast majority of cases the axillary glands were enlarged, and in about 75 per cent. were tubercular. In a number the supraclavicular glands were also enlarged. Spontaneous cure before suppuration must be exceedingly rare, and is not admitted by all to exist. Even after suppuration and discharge, through operation or by nature, few recorded cases showed a true healing. The process continues by extension, involving new areas before the older have healed, and death more or less slowly advances when the disease is left to itself, particularly through the involvement of the thoracic viscera.

The examination of the breast in this confluent form finds it firmer than normal, and this firmness is as a solid mass. Sections show an irregular cavity, or several cavities usually communicating, filled with turbid serum, detritus, or puriform liquid. The walls are dense, usually roughened with fringes and villosities. The lining is a soft, grayish or purplish membrane. Externally are found fibrous prolongations into the adjacent tissue. The tissue surrounding these cavities is of increased density and shows small grayish transparent areas, or yellowish opaque spots, representing the extension and infiltration of the tubercular process. Fistulae may connect the cavities with the exterior.

Roux's third form—the intraglandular cold abscess—is a terminal product, usually of the confluent tuberculosis, and is of slow development. I could find no record of its occurrence in a patient under nineteen years of age (Roux's case[62]). It occurred more frequently after pregnancy. Diminution in the size of the breast results. The abscesses are lined by a soft, reddish membrane, and contain a thin pus often with thicker masses of grayish or yellowish material. Tubercles and tubercle bacilli are, as a rule, not found in the walls or contents of these abscesses.

Miliary tuberculosis affecting the breasts has been found at autopsy.

In several cases the earlier symptoms and course of the mammary involvement varied from the usual form. In Orthmann's[63] (frequently quoted) it began apparently subcutaneously and resembled an ordinary furuncle. The base became indurated and extended, forming a tumor of considerable size. In Poirier's[64] two small vesiculated pimples with indurated base first appeared. In Kramer's[65] an ulcer appeared near the nipple, and later nodules formed within the breast and were connected to the ulcer by a band of indurated tissue. In Demme's[66] case induration began about the origin of a preformed sinus, the result of a simple mastitis.

In three cases retraction of the nipple was the first symptom noted. In Dubrueil's[67] two years before the tumor was discovered; in Verneuil's[68] the retraction began shortly after confinement, five years later a mass was discovered; in Warden's[69] it was noticed by the patient while carrying her third child, and eleven months after its birth, the mammary tumor was found.

In many cases the disease was regarded as primary in the glands and secondarily infecting the breast. A case of Reverdin's[70] is regarded as a proven example of this. Also eight of a series of thirty-seven collected by Berchold,[71] and Smith's[72] case, referred to above. Verneuil regards this as the usual method of infection, notwithstanding the known difference in the speed of progress of tubercular disease in the breast and lymphatic glands. Halstead and Le Count lay stress upon this retrogressive lymphatic involvement from the axillary glands and thoracic cavity, and believe that "a primary mammary gland tuberculosis has yet to be confirmed by autopsy." A hematogenous origin, however, is not to be ignored, while infection by way of the ducts is scarcely probable. Powers regards the infection in the breast as probably transmitted from the axillary lymphatics.

Auto-infection, by way of the blood current, from a previously existing focus of disease is probably the usual way.

The scarcity of the tubercle bacilli in the exudates and tissues is generally remarked. Gautier found that they were demonstrated but twenty-two times in seventy-seven cases collected. Scudder but twenty-nine times in eighty cases. Habermaas found two bacilli in twelve sections, Piskacek, a few only in 400 preparations. There are other observers who have failed to find them in over 100 sections. In a number of instances the more delicate inoculation test was positive, where they could not be found in sections.

In the cases of more recent years, when a greater number have been examined for the bacilli and inoculations oftener made, the percentage of positive results has been much higher.

The diagnosis of tuberculosis of the breast, in its earlier stages, cannot be made from the clinical symptoms and macroscopic appearance. This is particularly true of the discrete form and when enlarged glands or evidences of a tubercular focus elsewhere are lacking. If the disease is well advanced, or tuberculosis exists or has existed in some other part, there is less difficulty. In the majority of cases the diagnosis was not made prior to the operation.

The tumor may be mistaken for: (1) Simple Cysts, (2) Fibro-adenomata, (3) Carcinoma, (4) Sarcoma, (5) Gummata, (6) Actinomycosis. It may also be necessary to differentiate the process from a chronic interstitial mastitis.

Simple cysts are usually more circumscribed, fluctuate earlier, are painless, and there are no axillary glands. Aspiration demonstrates their character. With the fibro-adenomata there will be rather greater difficulty. They are apt to occur in young adults and are of slow growth. The fibromata are commoner, usually more distinctly movable in the gland, and the axilla is free. In Davis's case it is of interest to note that the tuberculosis had apparently been engrafted upon a pre-existing adenomatous tumor. In the earlier stages of carcinomatous disease there may be confusion. This trouble begins, however, as a rule, at a more advanced age, is usually more rapid and with greater pain. The axillary glands do not suppurate and are not painful at so early a date. Pilliet and Platet[73] and Warthin[74] have reported cases in which carcinoma and tuberculosis existed in the same breast. In sarcoma the growth is rapid and the skin is involved early. The cutaneous veins are apt to be enlarged. Gummata are to be distinguished by the history, coincident lesions, or response to specific treatment. The nodular form of gumma is rarer than the diffuse mastitis of syphilitic disease. In actinomycosis the axillary glands are usually not enlarged. The yellowish-green granulations characteristic of the fungus are formed. In chronic mastitis there is lack of glandular infiltration in the axilla. The feel is less that of a mass and more that of a spread out or diffuse process. In a case that came to me in the out-patient service at the hospital, it seemed necessary to excise a small bit of the tissue under cocaine to make a certain diagnosis.

The treatment of most accessible foci of tubercular disease at the present time is that of removal, with the institution of suitable climatic and hygienic conditions as soon thereafter as possible. Removal of the breast and axillary glands in extensive involvement is the rational method of treatment and offers the best hope for speedy and permanent cure. It is usually unnecessary to include the pectoral muscles. In primary cases and in those with the disease confined to the breast and axillary glands, the prognosis after breast amputation is good. Those of the so-called "tubercular disposition" will have a rather less favorable outlook for the future.

In secondary cases the prognosis depends upon the seat and degree of the primary lesion. Occasionally this will be of sufficient severity to contraindicate radical interference.

In the discrete or nodular form, a removal of the nodule together with a wedge-shaped section of the breast has been recommended, provided the case can be kept under observation for some time. There are those who prefer to leave the axillary glands undisturbed if they are not appreciably enlarged. Partial operations, however, are not entirely free from risk, although often possible and attractive. Lane's first case is an example of the danger of incomplete operation. The tubercular process and invasion usually extend beyond the point at which the eye ceases to appreciate it.

In the less common cases of cold abscess without changes in the axillary glands or where radical operation is contraindicated, aspiration or incision of the abscess with the injection of iodoform emulsion or of iodine and potassium iodide (Durante's method) has been recommended.

The following methods of treatment have been employed in the cases reported:

(1) Curetting of sinuses.
(2) Cauterization of sinuses.
(3) Injection of sinuses and cavities.
(4) Incision or aspiration of abscesses.
(5) Removal of the tumor alone.
(6) Removal of the axillary glands alone.
(7) Removal of the tumor and a portion of the breast.
(8) Removal of the breast and tumor.
(9) Removal of the breast and axillary glands.

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MESENTERIC THROMBOSIS, OPERATION—RECOVERY.

W. Scott Schley, M. D.

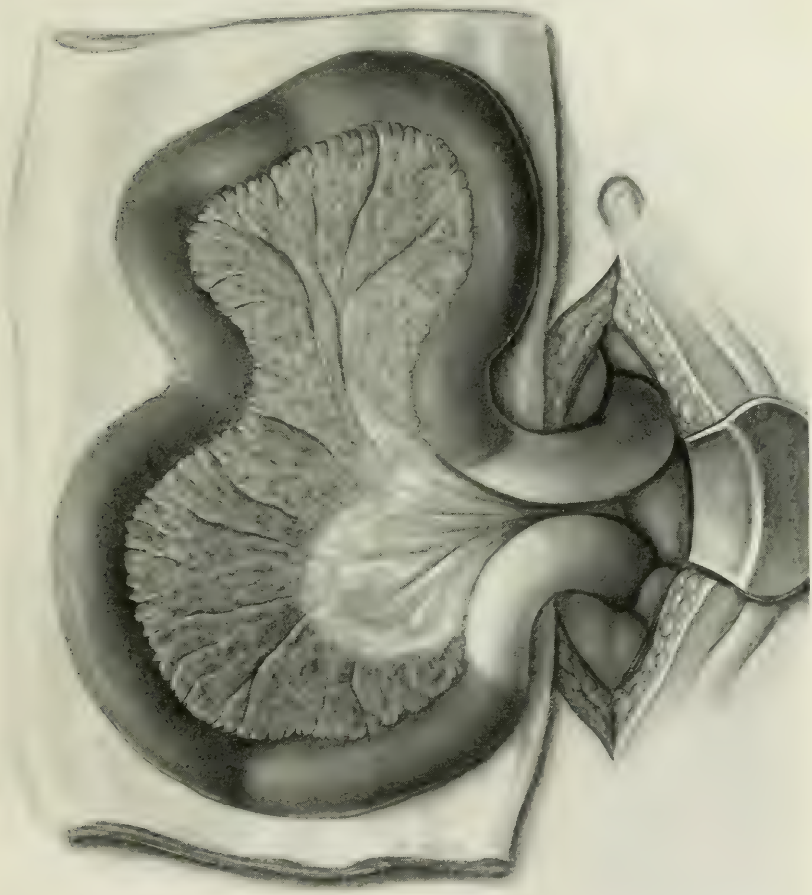
Surgical No. 83,888. G. K., a man of 42 years and bricklayer by occupation was admitted to the First Surgical Division, December 23rd, 1910, having been transferred from the Medical Division and the service of Dr. Janeway.

He had been taken sick twenty-four hours before entrance and several hours after a meal, with a sudden "sharp pain" across the upper abdomen. The pain was continuous from the onset and frequently radiated to the lower abdomen in a stab-like manner. He vomited once, two hours after the beginning of the attack. Vomitus contained no blood. Bowels moved normally morning before attack, but not afterwards, nor had he passed flatus. Urination normal prior to attack. No history of stomach, intestinal, rectal or urinary disturbances before attack. No venereal history could be obtained or history of abdominal trauma. He had had lobar pneumonia four years before, and but for that has worked for many years at his trade and says he has always been a strong healthy man, taking only an occasional glass of beer.

His temperature on admission was $100\frac{3}{5}$; pulse 94 and respiration 24; leucocyte count, 20,000, with a polynuclear percentage of 89.

Examination showed only a moderately distended abdomen with general rigidity. Tenderness to pressure was not marked and seemed somewhat greater over the upper half. Probably some fluid accumulation. The man had the appearance of suffering, and the action of one acutely ill, but as yet in good condition. No definite diagnosis could be made, but abdominal section was clearly indicated.

Operation (Dr. Schley). Ether anesthesia. On operating peritoneum in median line below the umbilicus about 200 c. c. of dark red, fairly clear serum escaped. It had no fecal odor. The intestines were moderately distended and showed slight general vascular engorgement. Just below the lower end of the incision, a deep black-



Mesenteric Thrombosis of venous type. Terminal loops of venous arches affected. Line of demarcation between necrosed and healthy mesentery very marked.

red loop of gut presented. It was found to be part of a good sized coil in the same condition. The gut was thickened and indurated, and showed a sharp line of demarcation at either end from the healthy intestine. There was a very narrow, quite injected area between the two. The mesentery was a deep chocolate brown with gray black mottling, and was involved for from two to six inches from attachment to gut; the thrombosis affecting the terminal arches. It was much thickened and indurated and almost black on section.

Bleeding from the arteries could be demonstrated, but the veins were thrombosed. The gut was excised four inches on either side of the involved area and the mesentery well beyond demarked lines and to where both vessels bled. The affected gut, nearly two feet in length, was later found to be filled with brownish, bloody fluid and detritus. There was no ulceration of the mucosa apparent to the eye. A rapid examination of the contiguous gut revealed nothing. The ends were brought together in this case by button, reinforced by Cushing stitch and the mesentery by continuous catgut, care being taken in suturings to include no vessel of size. The peritoneum was flushed with hot saline and abdomen closed by layer suture.

The patient stood the operative work well, and on the third day following, gas and some fecal matter began to come away in the rectal saline irrigations. After wound healing appeared primary in every respect, a moderate temperature elevation was followed by an opening in the central part of the scar discharging some pus and fecal matter. The latter continued fairly profusely for ten days. The patient steadily gained, however, and the fistula contracted.

At his discharge on February 14th, there remained a fine probe opening only, and dressings remained unsoiled for two or three days at a time. The button has not come away and radiographs show it still in the lower part of the ileum. The man is steadily gaining and further interference seems unwise at present. It is to be regretted that no cultures were taken from the thrombosed veins before the specimen was put in preservative.

It is of interest in connection with this case to summarize briefly some of the collected autopsy records and reports of this condition.

So far the malady in the acute form has proven almost universally fatal, but delayed operations and faulty technique have undoubtedly been partly responsible. Some fifteen autopsy records, at least, show a comparatively small area of involvement with nothing apparent in the recorded findings to contraindicate resection. Some of these would probably have been saved.

In the collection of 214 cases of Mesenteric Embolism and Thrombosis by Jackson, Porter and Quinby, 47 were operated, with 92% mortality. They consider the cases of the disease may be divided into two groups; acute and chronic; acute, those of sudden onset and by far the larger number and in which death occurs in a few hours or days; the chronic, formed by the small number of cases having an insidious onset, sometimes remitting symptoms, those with no symptoms referable to the abdomen during life and those in whom spontaneous recovery resulted. In either group arterial or venous closure may be the cause. There were 14 cases, equally divided between arterial and venous occlusion, that ran a course of over two months. They were apparently due to a thrombosis wtih intermittent progression and the establishment of a competent collateral circulation in the meantime. Four cases were found in 1600 post mortems at the Johns-Hopkins Hospital in which hemorrhagic infarction of the intestine had occurred without being suspected during life, collateral circulation having been established. Thrombosis of the larger mesenteric vessels and inferior cava has occurred without lethal termination. Brewer has recently reported a case of occlusion of the portal vein. On the other hand, in the acute cases, an inch of involved gut has proved fatal. Very few cases were diagnosed during life and in the majority the condition was only disclosed at the post mortem table.

The prompt recognition of a serious intra-abdominal condition in this case and the speedy transfer and operation were certainly very large factors in saving this man's life.

CONGENITAL INTESTINAL OCCLUSION.

JOHN DOUGLAS, M.D.

Patient A. L., age 36 hours. Admitted to Surgical Division B. October 16, 1909. The history was one of persistent repeated vomiting and absolute constipation since birth. The vomitus was stained with meconium. The physician who had delivered the child said that he had removed a greyish mass the size of a hazel nut, and resembling a phosphatic calculus, from the rectum before sending the patient to the hospital.

Examination showed marked abdominal distention, particularly in the epigastric region. There was no abdominal rigidity or elevation of temperature, but the pulse was very rapid. Rectal examination showed that no meconium was present. A diagnosis of intestinal obstruction was made and operation determined upon.

Operation by Dr. Douglas. After gastric lavage, an incision was made in the median line about one inch above and one inch below the umbilicus. Exploration showed that the small intestine which was greatly distended, ended in a blind pouch in the region of the umbilicus, the distance along the intestine from the pylorus being 63 cm. From the end of this pouch a mass of soft connective tissue in which were many fibrous bands, ran up to the umbilicus. Within this mass of connective tissue, two distinct segments of undeveloped intestine closed at both ends could be made out, one empty and one filled with soft material. From the same mass ran the continuation of the small intestine, undeveloped and undistended being only 7 mm. in diameter. The colon was 9 mm. and the sigmoid 5 mm. in diameter, while the distended small intestine was 2.5 cm. in diameter.

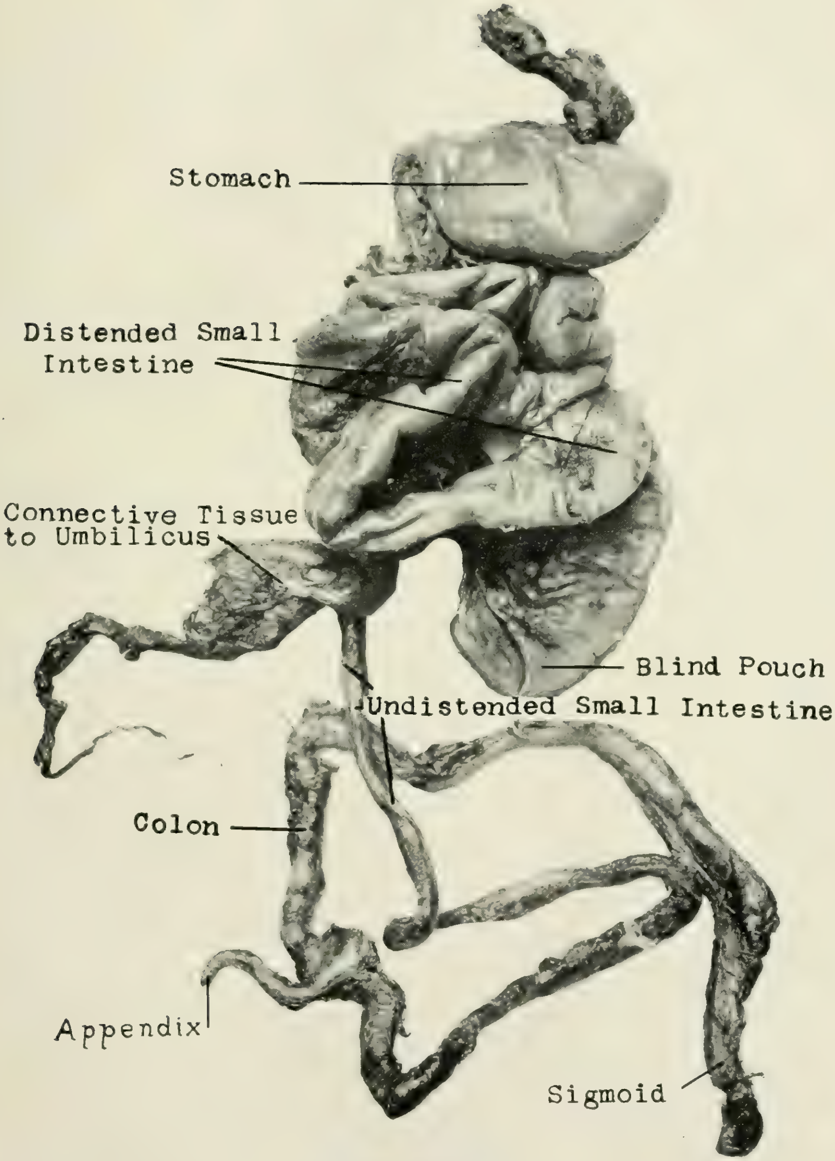
The obstruction was evidently due to lack of development caused by pressure of the remains of the omphalomesenteric duct or vessels, and the only possible treatment was enterostomy or an attempt at enteroenterostomy neither with much promise of success. While determining which course would be best to pursue, the child vomited a large amount of greyish fluid (notwithstanding the previous lavage) and inspired a quantity of the vomitus. Its condition became so bad that the abdomen was closed without further treatment, and the child died about four hours later.

Various conditions have been suggested or demonstrated as the cause of congenital intestinal obstruction. Foetal peritonitis, tuberculosis, syphilis, persistence of the ductus omphalo-mesentericus, ulceration with scar formation and contraction, foetal intussusception, compression by congenital tumors, anomalies of circulation with resulting ischaemia, and obliterating endarteritis. Kreuter considers all cases to be due to retardation of development. Stenosis or atresia may occur anywhere in the intestine either singly or in two or more areas. It is most frequent in the small intestine, next in the duodenum and least frequent in the large intestine.

Tuffier published the reports of 32 operations for this condition, 26 enterostomies, 4 enteroanastomoses, and 2 perineal ileostomies. All resulted fatally. Bossouski reported two cases of his own and 31 from the literature all of which were fatal. Since then Spriggs has reported 7 additional cases with the same result. Lane has performed gastroenterostomy for duodenal obstruction unsuccessfully.

The difficulty of performing an enteroenterostomy between the distended and the undeveloped portions of the intestine is great; and the small chance of keeping up the child nourishment with an enterostomy, especially if the obstruction be in the small intestine, does not hold out much hope of lessening the 100 per cent mortality. Spriggs recommends enterostomy with subsequent anastomosis. Clogg has suggested the attempt at dilatation of the undeveloped intestine with water pressure, at the time of operation.

While there has so far been no successful case reported, it is suggested, that in a favorable case in which only one obstruction is present below which the intestine is undistended, that the best chance of attaining success would be by doing a rapid enterostomy bringing both the distended and undistended ends of the intestine into the wound. Then if the child's condition improved in a day or so an attempt might be made to dilate with saline solution under pressure the undeveloped or undistended intestine with the hope of subsequently performing an enterostomy if the dilatation were successful. Whether this can be done or not remains to be proven.


Stomach
Distended Small Intestine
Connective Tissue to Umbilicus
Blind Pouch
Undistended Small Intestine
Colon
Appendix
Sigmoid

Congenital Intestinal Occlusion

MULTIPLE ABSCESSES OF ONE KIDNEY IN A CHILD.

John Douglas, M.D.

Patient R. K. No. 83279—Age 7. Admitted to Surgical Division B. November 15, 1910. Family History—negative. Past History. Always well up to 3 months ago, since which time the child has been ailing. She complained of headache, general lassitude, occasional slight fever and loss of appetite and weight. For the past month she has had pain in the right side on climbing stairs or on unusual muscular exertion, and has been very quiet. She has complained at times of pain on urination for several weeks, but has never passed any gravel, stones or blood. The urine was occasionally cloudy.

Present Illness. Two days before admission during the night, she began to vomit and have severe abdominal pain. She vomited all night and the following morning had a chill lasting 15 minutes followed by a high fever. The bowels moved twice that day and once the day following influenced by glycerine suppositories, prescribed by a physician, who had been called to attend the child. There was no blood in the stools.

On admission, the child looked sick. The temperature was $103\frac{4}{5}$ F, pulse 144 and respiration 40. There was a high total and polynuclear leucocyte count. Abdominal examination showed a tender mass on the right side slightly above the level of the umbilicus and there was moderated abdominal rigidity on the right side. Percussion elicited a dull tympany over the mass.

The child was admitted as an emergency case during the evening and the details in the history pointing toward a kidney involvement were not obtained until the following day when the parents returned to the hospital. It was believed that she had a high retrocaecal appendicular abscess, although a suppurative kidney lesion was considered. Immediate operation was advised.

Operation by Dr. Douglas. A high intermuscular incision was made over the mass, and on opening the peritoneum the appendix, markedly thickened, presented in the wound. This was rapidly removed by the purse string method and then the mass examined. This proved to be the right kidney, which was between two and three times the normal size and was studded with necrotic abscesses varying in size from a pin head to a centimeter in diameter. There were more than 40 of these abscesses. In addition, the pelvis of the kidney was distended and held about 100 cc. of cloudy urine. A nephrectomy was imperative and as the child's condition was rapidly becoming critical it appeared that there was less danger of removing the kidney by the abdominal route than if the abdominal incision were closed and a lumbar incision, requiring additional time, were made. The kidney was therefore removed through the original incision and a rubber dam drain inserted.

The temperature fell rapidly after the operation, the urine became normal, there was no subsequent infection of the wound and the child was discharged cured at the end of three weeks.

This case of multiple infection of one kidney is of interest on account of the age of the patient, and on account of possible hematogenous infection of the kidney occurring in a child who had not previously had some acute infection as an etiological factor. The previous history of the case is also of interest as it would seem to indicate that there might have been a displacement of the kidney with perhaps intermittent hydronephrosis, the displaced kidney being in a position where it was subject to traumatism, which has been shown by Brewer by his animal experiments, to be an etiological factor in acute hematogenous infection of the kidney.

On the other hand, while the macroscopical appearance of the specimen, showing numerous hemorrhagic areas studded with miliary abscesses, these areas being wedge shaped on section and looking like infarcts, would seem to indicate a hematogenous infection. The dilated kidney pelvis, together with the previous history of a cystitis, fever, malaise and lumbar pain, would point to the probability of the lesion being the result or a development of one of those cases of pyelitis due to colon bacillus infection, which are not uncommon in children, and which usually promptly subside under treatment with urotropin.

After nephrectomy the urine rapidly became normal. Unfortunately the desperate condition of the child during operation prevented time being taken to obtain a culture, and pathological examination of the specimen has failed to determine the route of the infection, whether ascending or hematogenous.

CYSTOSCOPY AS AN AID IN DIAGNOSIS.

From the Service of Dr. Robert Abbe and Dr. Walton Martin.

Henry G. Bugbee, M.D.

During the past six months it has been my privilege to make cystoscopic examinations on patients entering Dr. Abbe's Division, who presented symptoms referable to the genito-urinary tract. These patients have been under the care of Dr. Abbe and Dr. Walton Martin. It is through their courtesy that I am privileged to report them.

The object of this article is to show the value of cystoscopy and ureteral catheterization as an aid to diagnosis of lesions of the genito-urinary tract; also its value in eliminating this tract as a cause for general symptoms. When disease can be localized by cystoscopy, it can be studied, the advisability of operative interference decided and often the method of operating outlined.

To say that the instrument is infallible would be incorrect, for as in all surgical procedures, it presents certain disappointments. The shortcomings are gradually eliminated as one becomes more expert with the handling of the cystoscope, and the interpretations are more exact as one sees a larger number of pathological conditions. Often a needless operation is spared the patient, or it is found that a simple operative procedure will give complete relief.

In the diagnosis of inflammatory conditions of the bladder, especially tuberculosis; of new growths, malformations, and foreign bodies in the bladder; of hypertrophy of the prostate; the presence of calculi in the urinary tract; obstruction of the ureters; disease and new growths of the kidneys, more data can be obtained than from any other one method of diagnosis. Even though the character of the lesion is absolutely certain, from the history and physical signs, the functional capacity of the kidneys can only be ascertained by a separation of the urine from either kidney. This functionating capacity of the kidneys should be known before deciding upon any operative procedure upon either organ.

Cystoscopy and ureteral catheterization can be carried out in the majority of cases with slight discomfort to the patient. If the instrument can be introduced and the bladder distended with a medium which can be maintained clear for a long enough period of time, cystoscopy can be performed. When pathological lesions are present which render the slightest manipulation and distention of the organ painful to the patient, it may be advisable to use a local or general anesthetic. This, however, is seldom necessary.

The points to be noted in making a cystoscopic examination of any bladder are: the condition of the mucous membrane as regards its color, smoothness and glistening appearance; the characteristics of the blood-vessels; the appearance of the internal vesical sphincter and trigone (noting especially any irregularities indicating prostatic enlargement). The ureteral orifices should be carefully studied as regards position, size, shape, functionating power, the character of the urine emitted from each, volume of the swirl, rapidity of the secretion, and the force of contraction of the ureteral duct.

It is necessary to pass catheters to the kidneys only when an obstruction of the ureter is suspected, or it is desired to outline their course with the X-rays. Otherwise the catheters are advanced but a few centimeters along the ureters and the urine collected for analysis. The discomfort following the examination is slight and the danger of infection practically nil.

The cases reported represent those which come into the hands of any general surgeon. In every case the cystoscopic examination has proved of value. Only the more important facts are mentioned in the histories, physical examinations, cystoscopic examinations and operative reports. In several instances operative interference has been found to be unnecessary after the cystoscopic findings have been taken into consideration; in others the findings at operation verified those of the cystoscopic examination.

The cuts shown are from sketches of the conditions found, and represent fairly typical pictures of the lesions.

The first case was of a man, 63 years of age, married, who presented bladder symptoms extending over a period of five years. He denied venereal disease and up to that time he had enjoyed good health. During the past five years he had suffered from frequency, burning, dribbling and at times inability to empty the bladder. This had necessitated the use of the catheter with the consequent cystitis, which was present on his admission to the hospital. Aside from the urinary disturbance he was in fairly good condition; he had lost little weight and had been up and about.

The general physical examination on admission, May 9, 1910, was negative, with the exception of a moderate general enlargement of the prostate gland. The bladder contained 16 ounces of ammoniacal, cloudy, residual urine. The cystoscopic examination was of interest, in that it showed a collar of prostatic tissue surrounding the vesical neck and the changes in the bladder wall which accompany such a condition. These changes consisted in chronic inflammation of the mucous membrane, shown by its dull, gray color, uneven surface covered with flakes of epithelium and mucus; trabeculation, with the formation of numerous diverticula, and dilatation of the ureteral orifices. These orifices functionated normally and the swirl of clear urine coming from each showed that the pathological condition was localized in the lower urinary tract. The prostate gland posterior to the vesical neck was prominent, and seemed to be distinctly raised above the surrounding bladder wall.

At the operation by Dr. Abbe the prostatic enlargement was found to consist mainly of a hard fibrous ring surrounding the vesical neck, which was with some difficulty dilated, but was not sufficiently large to necessitate removal.

The cystoscopic examination revealed the prostatic obstruction, but the prominence of the gland posteriorly was probably due to the falling away of the bladder wall into a pouch behind it, caused by its complete atony.

Because of the negative findings the cystoscope was of value in the second case. This man was 40 years of age, married, an Englishman, in good general health. Aside from an attack of gall-stones 16 years before and appendicitis 6 years before, he had been well until two months prior to admission to the hospital when he was seized with a sudden severe pain in the left side of the abdomen. This pain radiated to the testicle and penis. He had a chill, followed by slight fever. The pain disappeared, but was superseded by a dull ache which has persisted. There had been several exacerbations of the pain, all similar to the first attack. No urinary disturbance.

Other than a small hernia in the appendectomy scar, the physical examination threw no light on the condition.

By cystoscopic examination and differentiation of the urine from either kidney, the possibility of a calculus obstruction of the ureter was eliminated.

At the operation by Dr. Abbe for radical cure of the hernia, an exploration confirmed a normal condition of the kidneys and ureters. A loop of intestine was found adherent to the liver, giving a partial obstruction. This was freed and the patient, after the operation, was relieved of the old pain.

In the third patient an operation which probably would have left the patient in a more deplorable state, was prevented by the cystoscopy. This man, 52 years of age, had suffered from frequency, burning and dribbling for seven months. The urine had been bloody. He had lost much weight and on his admission, August 17, 1910, he was considerably emaciated.

His physical examination revealed a hard mass above the symphysis pubis, which was apparently connected with the large prostate to be felt on rectal examination.

The cystoscopic examination revealed a carcinoma, involving the prostate and about two-thirds of the bladder wall. The growth showed the various stages of degeneration, from papillary formations to deep ulcerations (see Fig. I., frontispiece). The slightest motion of the cystoscope in the bladder caused bleeding from the ulcerated surface. The condition was beyond all operative relief.

A man, 53 years of age, who entered the hospital, August 14, 1910, with a swollen testicle, undoubtedly tubercular, presented an opportunity for determining the extent of the invasion present. Inasmuch as the greater number of tubercular infections of the genito-urinary tract are descending, arising in the kidneys from a hematogenous infection, this was most essential to determine. The physical examination of the patient gave evidence of no lesions in other parts of the body and the cystoscopic examination, with analysis of differentiated urines, revealed a normal bladder and healthy, functionating kidneys. The testicle was removed by Dr. Walton Martin, found to be a sac containing thick pus, later verified as tubercular by pathological examination.

It was possible, due to the cystoscopic examination, to give this patient a good prognosis.

Another patient, a male, Italian, 38 years of age, gave a history of the removal of a vesical calculus 7 years ago. One week before entering the hospital he was seized with a sharp attack of colic in the right lumbar region, the pain radiating to the groin and testicle of the same side. He passed a small stone in some bloody urine. This was followed by a similar attack on the following day and 1 cc. of phosphatic sediment was found in the urine. Three days before admission, August 15, 1910, he had a third attack. No stone was passed at this time and the pain continued to the date of admission.

The physical examination revealed tenderness of a slightly enlarged right kidney; also tenderness along the course of the right ureter.

The cystoscopy is of extreme interest as showing some of the possibilities of the instrument from an operative standpoint.

The filling of the bladder caused slight tenesmus. Examination showed a marked congestion of the trigone and vesical neck and dilatation of the blood-vessels. The left ureteral orifice was normal and the stream of urine emitted from it was clear. About the right ureteral orifice for a distance of 2 cm. was a deep red blush, simulating an early tuberculosis.

The orifice was dilated and golf-holed in appearance, as shown in Fig. II. This dilatation of the orifice would not be present in an early tuberculosis, but is indicative of calculus lodged in the ureters. A catheter entered the ureter, but was arrested 1 cm. from the bladder. By manipulating the catheter, a stone, the size of a pea, was dislodged and discharged into the bladder, followed by a gush of urine. The catheter now passed to the kidney. Urine discharged was at first bloody, but later clear. The stone was evacuated from the bladder. The patient stated that he experienced immediate relief on the discharge of the stone from the ureter.

The next patient, a man of 44, Italian, entered the hospital, September 28, 1910, complaining of frequent urination, accompanied by tenesmus and burning in the bladder neck, these symptoms extending over a period of 1 year. He had lost 25 pounds in weight, but was in fairly good physical condition. The prostate gland was markedly enlarged, as shown by rectal examination.

Cystoscopic examination verified this fact, showing also a well marked median prostatic lobe. The picture of obstruction to the vesical outlet was present, as shown by the chronic cystitis, trabeculation and sacculation of the bladder wall, and dilatation of the ureteral orifices. Analysis of the differentiated urines showed a healthy condition of the kidneys.

Perineal prostatectomy by Dr. Martin verified the cystoscopic findings.

The value of the cystoscopic examination as a preliminary to operation is shown in the next case. This man, 63 years of age, gave symptoms of prostatic obstruction, consisting in frequency of urination day and night, burning, tenesmus and occasional retention.

The physical examination was negative, aside from an extensive enlargement of the prostate, to be felt by rectum.

Cystoscopy revealed, aside from a remarkably hypertrophied gland, with the signs of vesical obstruction, *i. e.*, chronic cystitis, trabeculation, diverticula and dilatation of the ureteral orifices, a vesical calculus which was nearly obscured by the prostate. The calculus was lodged in a sacculation and covered with pus and mucus (see Fig. III). The presence of stone had given no symptoms and it could easily have been overlooked when doing the prostatectomy.

The gland and stone were removed by the perineal route by Dr. Martin. The prostate showed general hypertrophy. The calculus was the size of a large walnut.

The next patient examined gave a similar history and the findings were almost identical. He was 69 years of age, had noticed frequency of urination for several years. The stream was started with difficulty and he dribbled at night. The stream was small, and at times he could not void, the catheter being required. Had lost some weight, but the physical examination showed a well developed and fairly well nourished man. Aside from the enlargement of the prostate, to be felt per rectum, no pathological condition could be defined.

Cystoscopic examination, September 14, 1910, revealed an enlargement of the prostate which involved the entire gland. The outline of the lateral lobes, also a median lobe, was distinct. The bladder wall about the base and vesical neck was of a deep red color. The remainder of the bladder mucous membrane was somewhat congested, and trabeculation with the formation of diverticula in the bladder wall was well marked. Resting on the raised prostate, at the base of the bladder, a calculus the size of an English walnut could be distinctly seen. It was rough, gray and partially covered with mucus, as shown in Fig. IV. The ureteral orifices were dilated, but functionated normally and the swirl of urine from each was clear. Dr. Martin performed a perineal prostatectomy on the patient, as in the preceding one. The stone was removed at the same time. This stone, by dropping back into the fundus at the time of operation, by being covered with pus, and thus not easily felt, might have been overlooked, had it not been found with the cystoscope.

The following case serves to illustrate the use of the cystoscope as a means to eliminate the genito-urinary tract from being a causative factor in the production of certain symptoms possibly referable to it, at least from a surgical aspect.

A female, 23 years of age, admitted September 27th, suffering from pain in the left lumbar region. This pain had come on following an attack of uremia. It was dull in character, localized in this region and accompanied by no urinary symptoms.

Physical examination was negative and cystoscopy and differentiation of the urine showed hemorrhagic nephritis.

An interesting case was presented in that of a man 49 years of age. He gave a history on admission, October 4, 1910, that two years ago he had suffered from hematuria and at the time calcium oxalate crystals were passed in the urine. From that time he had been well until two weeks before admission. On admission he had had a severe pain in the left side and the back which radiated in all directions. It was not modified by posture, lasted one half an hour, and was followed by hematuria. A few hours later he had a similar attack, but less severe, which was relieved by morphine. Since then he had had a dull ache in the left side of the abdomen, but no sharp attacks.

His general health was excellent and physical examination negative.

The cystoscopic examination revealed a normal condition of the mucous membrane of the fundus and lateral walls, marked congestion of the mucous membrane of the trigone and base. The trigone was raised by an enlargement of the prostate. The ureteral openings were located on either side of this prominence. They each functionated normally and the urine emitted was clear. Catheters entered the orifices with some difficulty, owing to the distortion of the ureteral ducts caused by the prostatic enlargement. The flow of urine from each catheter, however, was free.

Although the clinical history pointed to nephrolithiasis, the cystoscopic findings tended to discredit it. X-ray examinations agreed with the cystoscopic findings.

A male, 26 years of age, entering October 14, 1910, gave a history of 3 years ago having an attack of severe pain in the right lumbar region, radiating upward under the ribs and down to the scrotum. A similar attack had seized him 6 weeks before admission and he then had a dull ache in the side, which had persisted. Occasional attacks of lesser severity had occurred, during which he had vomited. No urinary symptoms. His general physical condition was good.

Cystoscopic examination and differentiation of the urine from either kidney showed a normal condition of this tract.

The last patient examined was a most interesting one. Again the cystoscopic examination made the diagnosis certain. This man, 44 years of age, admitted October 12, 1910, gave a history of burning and frequency of urination, beginning two months before entering the hospital. A sharp pain in the perineum was present while passing the last few drops of urine. Urination was frequent by night, as well as by day. There had been no macroscopic blood in the urine. Had lost 15 pounds.

Physical examination was negative and the case was thought to be one of prostatic obstruction.

Cystoscopic examination revealed a marked congestion of the mucous membrane of the trigone and vesical neck and a slight enlargement of the prostate gland. The left ureteral orifice was normal in appearance and the swirl of urine emitted was clear. The right ureteral orifice was hidden from view by a rough, gray calculus, the size of a walnut.

The calculus was removed by Dr. Martin.

Here again the symptoms caused by the presence of the stone could easily have been confused with those of prostatic irritation.

As stated at the beginning of the paper, these are not extraordinary cases, but represent types often seen. It seems justifiable to state that cystoscopy should be a part of the examination on all patients presenting symptoms referable to the genito-urinary tract.

Medical Service

MEDICAL STATISTICS FOR 1910

DISEASES DUE TO MICRO-ORGANISMS

INFECTIVE DISEASES

	Sex		Results				
	Male	Female	Cured	Improved	Unimproved	Died	Totals
Brill's disease	1	...	1	...	...	...	1
Cerebrospinal fever	...	1	...	...	...	1	1
Diphtheria	...	11	11	...	...	...	11
Dysentery (amoebic)	2	...	2	...	...	...	2
Erysipelas	...	1	...	...	1	...	1
Erysipelas, abscess of parotid gland	...	1	...	1	...	...	1
Erysipelas, eczema	...	1	...	1	...	...	1
Gonococcus endometritis, peritonitis	...	1	...	1	...	...	1
Influenza	6	38	43	1	...	...	44
Influenza, myocarditis	...	1	1	...	...	...	1
Malaria	5	2	7	...	...	...	7
Malaria, secondary anemia	...	1	...	1	...	...	1
Malaria, subacute articular rheumatism	1	...	...	1	...	...	1
Malaria (tertian)	...	2	2	...	...	...	2
Parotitis	2	...	1	1	...	...	2
Rheumatism (subacute articular)	2	10	5	7	...	...	12
Rheumatism (subac. art.), cervical adenitis	.	1	1	...	...	...	1
Rheumatism (subac. art.), myocardial degeneration	1	...	...	...	1	...	1
Rheumatism (acute articular)	10	8	16	2	...	...	18
Rheumatism (ac. art.), ac. appendicitis	1	...	1	...	...	...	1
Rheumatism (ac. art.), arthritis of elbow	...	1	...	1	...	...	1
Rheumatism (ac. art.), cystocele, rectocele, laceration of cervix and perineum, retroversion of uterus, prolapse of ovary	...	1	...	1	...	...	1
Rheumatism (ac. art.), erythema nodosum	1	...	...	1	...	...	1
Rheumatism (ac. art.), mitral and aortic insufficiency	3	2	2	3	...	...	5
Rheumatism (ac. art.), mitral stenosis and insufficiency	4	6	...	10	...	...	10
Rheumatism (ac. art.), mitral stenosis and insufficiency, arterio-sclerosis	1	...	...	1	...	...	1
Rheumatism (ac. art.), nephritis	1	...	...	1	...	...	1
Rheumatism (ac. art.), pericarditis, mitral insufficiency	1	1	...	2	...	...	2
Rheumatism (ac. art.), peripheral neuritis	1	...	...	1	...	...	1
Rheumatism (ac. art.), pleurisy with effusion, mitral insufficiency	1	...	...	1	...	...	1
Rheumatism (intercostal), mitral stenosis and insufficiency	1	...	...	1	...	...	1
Scarlet fever	...	3	2	...	1	...	3
Syphilis (primary), chancre of abdomen	1	...	...	1	...	...	1
Syphilis (primary), chancre of lip	1	...	...	1	...	...	1
Syphilis (secondary)	3	1	...	4	...	...	4
Syphilis (secondary,) eczema of foot, carbuncle of back	.	1	1	...	...	...	1
Syphilis (secondary), pharyngitis	1	...	...	1	...	...	1
Syphilis (tertiary)	4	1	...	4	1	...	5
Syphilis (tertiary), arterio-sclerosis, mitral and aortic insufficiency	1	...	...	...	...	1	1
Syphilis (tertiary), aortic and mitral insufficiency, dilatation of aorta with aneurism	1	...	...	...	...	1	1
Syphilis (tertiary), endarteritis, dilatation of aorta, sub-diaphragmmatic aneurism	1	...	...	1	...	...	1
Syphilis (tertiary), endarteritis, neuroretinitis	1	...	...	1	...	...	1
Syphilis (tertiary), gumma of brain, hemiplegia	...	1	...	1	...	...	1
Syphilis (tertiary), gumma of liver	1	1	...	2	...	...	2
Syphilis (tertiary), gumma of spinal cord	1	...	...	1	...	...	1
Syphilis (tertiary), nephritis, cirrhosis of liver	1	...	...	1	...	...	1
Syphilis (tertiary), orchitis, dactylitis	1	...	...	...	...	...	1
Syphilis (tertiary), ulcers of leg, bronchitis	1	...	...	1	...	...	1
Syphilis (cerebral), spastic paraplegia	1	...	...	1	...	...	1
Syphilis (cerebral), nephritis, uremia	1	...	...	...	...	1	1
Tuberculosis of lungs	33	44	...	49	10	18	77
Tbc. of lungs, coronary thrombosis, diabetes mellitus, arterio-sclerosis	1	...	...	...	...	1	1
Tbc. of lungs, delirium tremens	1	1	...	1	...	1	2

	Male	F.	C.	Imp.	Un.	D.	Tot.
Tbc. of lungs, fecal fistula	...	1	...	...	1	...	1
Tbc. of lungs, tbc. of hip	1	...	...	1	...	...	1
Tbc. of lungs, lobar pneumonia	1	1	...	1	...	1	2
Tbc. of lungs, mitral insufficiency, acute articular rheumatism	...	1	...	1	...	...	1
Tbc. of lungs, mitral stenosis and insufficiency	...	1	...	1	...	...	1
Tbc. of lungs, pleurisy with effusion	1	1	...	2	...	...	2
Tbc. of lungs pregnancy	...	1	...	1	...	...	1
Tbc. of lungs, pulmonary emphysema	1	...	...	1	...	...	1
Tbc. of lungs, prolapse of uterus	...	1	...	1	...	...	1
Tbc. of lungs, vomiting of pregnancy	...	1	...	1	...	...	1
Tbc. of lungs, tbc. enteritis	1	2	...	...	...	3	3
Tbc. of lungs, tbc. of glands of neck	...	2	...	2	...	...	2
Tbc. of lungs, tbc of peritoneum	1	...	...	...	1	...	1
Tbc. of lungs, tbc. of pleura	1	...	...	1	...	...	1
Tbc. of lungs, tbc. of larynx	2	1	...	2	1	...	3
Tbc. of lungs, tbc. meningitis	1	...	...	...	...	1	1
Tbc. of lungs, tbc. fistula in ano, tbc. enteritis	1	...	...	...	...	1	1
Tbc. of lungs (incipient)	...	1	...	1	...	...	1
Tbc. of peritoneal glands	1	...	...	...	...	1	1
Tbc. of peritoneum	2	...	...	2	...	...	2
Tbc. of pleura	1	3	1	3	...	...	4
Tbc. of spine	...	1	...	1	...	...	1
Tbc. of spine, retroversion of uterus, laceration of cervix and perineum, salpingitis	...	1	...	1	...	...	1
Tbc. meningitis	3	...	...	...	...	3	3
Tuberculosis (miliary)	...	1	...	...	...	1	1
Tuberculosis (miliary), gonococcus vaginitis	...	1	...	...	1	...	1
Tuberculosis (miliary), arthritis deformans	...	1	...	1	...	...	1
Typhoid fever	35	30	62	1	...	2	65
Typhoid fever with hemorrhages	3	3	3	...	...	3	6
Typhoid fever with perforation	1	...	...	...	...	1	1
Typhoid fever with relapse	2	2	4	...	...	...	4
Typhoid fever with typhoid spine	1	...	1	...	...	...	1
Typhoid fever, cholecystitis, tape-worm	...	1	...	1	...	...	1
Typhoid fever, lobar pneumonia, intestinal hemorrhage	...	1	...	...	...	1	1
Typhoid fever, lympho-sarcoma of sub-mental region	1	...	...	1	...	...	1
Typhoid fever, otitis media, mitral insufficiency	2	...	1	1	...	...	2
Typhoid fever, post-typhoid psychosis	...	1	...	1	...	...	1
Typhoid fever, terminal lobar pneumonia, laryngitis, hysteria	...	1	...	...	...	1	1
Typhoid fever, thrombo-phlebitis of popliteal space	...	1	...	1	...	...	1
Typhoid fever, thrombosis of femoral and popliteal arteries	1	...	1	...	...	...	1
Typhoid convalescence	1	...	1	...	...	...	1
Varicella	...	1	1	...	...	...	1
	166	206	171	141	16	44	372

LOCAL INFECTIONS

	Male	F.	C.	Imp.	Un.	D.	Tot.
Amoebic abscess of liver	1	...	...	...	1	...	1
Infected finger	1	1	2	...	...	...	2
Bacillus coli communis in kidney and bladder	1	...	1	...	...	...	1
Pneumococcus meningitis, septicemia, sinus thrombosis following mastoiditis	1	...	...	...	...	1	1
Stitch abscess	...	1	...	1	...	...	1
Streptococcus meningitis	1	...	...	...	...	1	1
	5	2	3	1	1	2	7

ALIMENTARY SYSTEM

INTESTINES

	Male	F.	C.	Imp.	Un.	D.	Tot.
Colitis	...	1	1	...	...	...	1
Colitis (ulcerative)	...	1	...	...	...	1	1
Constipation	...	2	...	2	...	...	2
Duodenal ulcer	1	...	...	1	...	...	1
Entero-colitis (chronic)	...	2	1	...	1	...	2
Entero-colitis (subacute), retroversion of uterus	...	1	...	1	...	...	1
Entero-colitis (acute)	2	1	3	...	...	...	3
Enteroptosis	...	3	...	3	...	...	3
Enteroptosis, gastric hyperacidity	...	1	...	1	...	...	1
Enteroptosis, pharyngitis	...	1	...	1	...	...	1
Gastroenteritis	2	1	3	...	...	...	3
Ileus	1	1	1	1	...	...	2
	6	15	9	10	1	1	21

LIVER

	Male	F.	C.	Imp.	Un.	D.	Tot.
Abscess of liver, rupture into lung	1	...	...	...	...	1	1
Cirrhosis of liver	1	...	...	1	...	...	1

	Male	F.	C.	Imp.	Un.	D.	Tot.
Liver—Cont.							
Cirrhosis of liver, ascites	1	...	...	1	...	...	1
Cirrhosis of liver, achylia gastrica	1	...	...	1	...	...	1
Cirrhosis of liver, alcoholic gastritis	1	...	...	1	...	...	1
Cirrhosis of liver, hematemesis, anemia	1	...	...	1	...	...	1
Cirrhosis of liver, chr. nephritis	1	...	...	1	...	...	1
Cirrhosis of liver, nephroptosis	...	1	...	1	...	...	1
Cirrhosis of liver, neurasthenia	1	...	...	1	...	...	1
	8	1	...	8	...	1	9
BILE PASSAGES							
Cholangitis (catarrhal)	2	1	2	...	1	...	3
Cholecystitis	1	...	1	...	...	...	1
Cholelithiasis	1	2	...	2	1	...	3
Cholelithiasis, hyperchlorhydria	1	...	...	1	...	...	1
Cholelithiasis, cholecystitis, hyperchlorhydria, neurasthenia	...	1	...	...	1	...	1
	5	4	3	3	3	...	19
MOUTH, TEETH AND GUMS							
Pyorrhea alveolaris	...	1	...	1	...	...	1
PANCREAS							
Calculus in pancreas, pancreatic diabetes	1	...	...	...	1	...	1
PERITONEUM, ETC.							
Ascites	...	2	...	2	...	...	2
Perinephritic abscess	1	...	...	...	1	...	1
Peritoneal adhesions, neurasthenia	...	1	...	1	...	...	1
	1	3	...	3	1	...	4
PHARYNX, TONSILS AND NASOPHARYNX							
Abscess (peritonsillar)	1	4	5	...	...	...	5
Abscess (peritonsillar), hypertrophied tonsils	...	1	...	1	...	...	1
Adenoids	...	1	1	...	...	...	1
Tonsillitis (chronic)	4	4	8	...	...	...	8
Tonsillitis (chronic), pharyngitis	...	1	1	...	...	...	1
Tonsillitis (chronic), hypertrophy of tonsils, mitral stenosis and insufficiency	...	1	...	1	...	...	1
Tonsillitis (follicular)	4	2	6	...	...	...	6
Tonsillitis (follicular), abscess of tongue	...	1	...	1	...	...	1
Tonsillitis (follicular), hypertrophy of tonsils	...	1	1	...	...	...	1
Tonsillitis (follicular), peritonsillar abscess	3	...	3	...	...	...	3
Tonsillitis (follicular), peritonsillar abscess, hypertrophy of tonsils	1	...	...	1	...	...	1
Tonsillitis (follicular), retroversion of uterus, floating kidney	...	1	...	1	...	...	1
	13	17	25	5	...	...	30
RECTUM							
Fistula in ano, starvation, inanition	1	...	...	1	...	...	1
Hemorrhoids (internal), secondary anemia	1	...	...	1	...	...	1
	2	...	...	2	...	...	2
STOMACH							
Dyspepsia	1	5	5	1	...	...	6
Dyspepsia (nervous)	2	1	...	3	...	...	3
Dyspepsia (nervous), insomnia	1	...	...	1	...	...	1
Gastritis (alcoholic)	4	...	...	4	...	...	4
Gastritis (alcoholic), multiple neuritis	...	1	...	1	...	...	1
Gastritis (alcoholic), multiple neuritis, cirrhosis of liver, tbc. of lungs	...	1	...	1	...	...	1
Gastritis (acute)	...	4	3	1	...	...	4
Gastritis (acute), nephritis	1	...	1	...	...	...	1
Gastritis (chronic)	5	5	1	6	3	...	10
Gastritis (chronic), congenital inguinal hernia	1	...	...	1	...	...	1
Gastritis (chronic), constipation	...	1	1	...	...	...	1
Hyperchlorhydria	3	4	...	6	1	...	7
Hyperchlorhydria, chr. gastritis	6	2	2	5	1	...	8
Hyperchlorhydria, chr. gastritis, duodenal ulcer	1	...	...	1	...	...	1
Hypochlorhydria, chr. gastritis	1	...	...	1	...	...	1
Stenosis of pylorus of stomach	2	2	...	3	1	...	4
Ulcer of stomach	4	4	7	1	...	...	8
	32	30	20	36	6	...	62

	Male	F.	C.	Imp.	Un.	D.	Tot.
VERMIFORM APPENDIX							
Acute appendicitis	1	...	1	...	...	...	1
Acute appendicitis with abscess	1	...	...	...	1	...	1
Acute gangrenous appendicitis with abscess	...	1	...	...	...	1	1
Chronic appendicitis	...	1	...	1	...	...	1
	2	2	1	1	1	1	4
CARDIO-VASCULAR SYSTEM							
BLOOD							
Anemia (pernicious)	1	3	...	3	...	1	4
Anemia (pernicious), lobar pneumonia, mitral stenosis and insufficiency	...	1	...	...	...	1	1
Anemia (secondary)	...	4	...	4	...	...	4
Anemia (secondary), hemoptysis	1	...	1	1	...	...	1
Anemia (secondary), tonsillitis	...	1	...	...	...	...	1
Anemia (secondary), uterine hemorrhage, septicemia	...	1	...	...	...	1	1
Chlorosis	...	1	...	1	...	...	1
Leukemia (lymphatic)	1	...	...	...	...	1	1
Leukemia (myelogenous)	1	1	...	...	2	...	2
Pseudoleukemia	1	2	...	...	2	1	3
Pseudoleukemia, hydrothorax	1	...	...	...	1	...	1
	6	14	1	9	5	5	20
ARTERIES							
Aneurysm of aorta	4	1	...	3	...	2	5
Aneurysm of aorta, mitral and aortic insufficiency	1	...	...	1	...	...	1
Aneurysm of common carotid	1	...	...	...	1	...	1
Arterio-sclerosis	1	...	...	1	...	...	1
Arterio-sclerosis, cardio-fibrosis, chr. nephritis, senility	1	...	...	...	...	1	1
Arterio-sclerosis, chr. nephritis, neurasthenia	...	1	...	1	...	...	1
Arterio-sclerosis, myocardial insufficiency, general anasarca, chr. nephritis	1	...	...	1	...	...	1
Atheroma of aorta, arterio-sclerosis, mitral and aortic insufficiency	1	...	...	1	...	...	1
Endarteritis (cerebral), cerebral thrombosis, terminal pneumonia	...	1	...	...	...	1	1
Thrombosis of coronary arteries	...	1	...	...	...	1	1
Thrombosis of mesenteric arteries	1	...	...	...	1	...	1
Thrombosis of axillary, external jugular and basilic veins, hydrothorax, mitral and aortic insufficiency	...	1	...	...	...	1	1
	11	5	...	8	2	6	16
VEINS							
Phlebitis of veins of leg	...	1	1	...	...	...	1
Thrombo-phlebitis of cavernous sinus, suppurative meningitis	1	...	...	...	...	1	1
	1	1	1	...	...	1	2
HEART							
Dilatation (acute), cirrhosis of liver, ascites	...	1	...	1	...	...	1
Endocarditis (septic)	1	2	...	...	...	3	3
Endocarditis (septic), pyelitis	...	1	...	...	...	1	1
Endocarditis (septic), mitral stenosis, aortic insufficiency, pulmonary infarct	...	1	...	...	...	1	1
Endocarditis (septic), mitral stenosis and insufficiency	1	...	...	...	1	...	1
Endocarditis (septic), nephritis, suppurative pleurisy, pulmonary infarct	1	...	...	...	...	1	1
Endocarditis (acute), partial decompensation, mitral insufficiency, chlorosis	...	1	...	1	...	...	1
Fatty heart, myocarditis	1	...	...	1	...	...	1
Heart block	1	...	...	1	...	...	1
Hypertrophy of heart, cervical adenitis	...	1	...	1	...	...	1
Myocardial degeneration, cirrhosis of liver, chr. nephritis	1	...	...	...	...	1	1
Myocardial degeneration with dilatation	1	...	...	...	...	1	1
Myocarditis	2	1	...	3	...	...	3
Myocarditis, mitral insufficiency	1	1	...	1	1	...	2
Myocarditis, chr. endocarditis, chr. nephritis	...	2	...	1	...	1	2
Myocarditis, double hydrothorax	1	...	...	1	...	...	1
Myocarditis, chr. nephritis	1	...	...	1	...	...	1
Valvular Diseases:							
Mitral insufficiency	3	2	...	3	...	2	5
Mitral insufficiency, myocarditis, chr. nephritis	...	1	...	...	...	1	1
Mitral insufficiency, myocarditis, arterio-sclerosis	1	...	...	...	...	1	1
Mitral insufficiency, arterio-sclerosis, emphysema, chr. nephritis	1	...	...	1	...	...	1
Mitral insufficiency, strangulated inguinal hernia	...	1	...	1	...	...	1
Mitral insufficiency and stenosis	1	5	...	3	...	3	6
Mitral insufficiency and stenosis, aortic insufficiency, chr. nephritis	...	1	...	1	...	...	1
Mitral insufficiency and stenosis, pulmonary infarct	...	1	...	1	...	...	1

	Male	F.	C.	Imp.	Un.	D.	Tot.
Heart—Cont.							
Mitral insufficiency and stenosis, pleurisy, neurasthenia	...	1	...	1	...	...	1
Mitral and aortic insufficiency	3	3	...	4	...	2	6
Mitral and tricuspid insufficiency	...	1	...	1	...	...	1
Mitral stenosis	1	2	...	2	1	...	3
Mitral stenosis and aortic insufficiency	...	1	...	...	...	1	1
Mitral regurgitation	...	1	...	...	...	1	1
Aortic insufficiency	4	1	...	2	...	3	5
Aortic insufficiency, arterio-sclerosis	2	...	...	2	...	...	2
	28	32	...	34	3	23	60
PERICARDIUM							
Adherent pericardium, mitral insufficiency, chr. nephritis	...	1	...	...	...	1	1
Adherent pericardium, aortic insufficiency and stenosis, mitral stenosis	.	1	...	1	...	...	1
Suppurative pericarditis, partial obstruction of colon, congestion of lungs, chr. nephritis, arterio-sclerosis	1	...	...	...	1	...	1
	1	2	...	1	1	1	3
LYMPH GLANDS							
Acute cervical lymphadenitis	1	...	...	1	...	...	1
CONNECTIVE TISSUE							
Abscess of thigh	...	1	...	1	...	...	1
Polyserositis	1	...	...	...	...	1	1
Oedema of ankles	1	...	...	1	...	...	1
	2	1	...	2	...	1	3
DUCTLESS GLANDS							
Goitre (simple), partial stenosis of trachea, ac. bronchitis	1	...	...	...	1	...	1
Goitre (fibrous)	...	1	...	...	...	1	1
Goitre (exophthalmic)	...	1	...	1	...	...	1
Goitre (exophthalmic), tbc. kidney	...	1	...	...	...	1	1
	1	3	...	1	1	2	4
MUSCULAR SYSTEM							
Chr. muscular rheumatism	2	...	...	1	1	...	2
NERVOUS SYSTEM							
BRAIN							
Apoplexy	1	1	...	2	...	...	2
Apoplexy, cerebral thrombosis, arterio-sclerosis	1	...	...	1	...	...	1
Apoplexy, hemiplegia, glycosuria	1	...	...	1	...	...	1
Hemiplegia	...	2	...	2	...	...	2
Hemorrhage with aphasia	1	...	...	...	...	1	1
Meningitis (basillar)	1	...	...	1	...	...	1
Paralysis (bulbar), anterior poliomyelitis	...	1	...	...	...	1	1
Oedema of brain, purpura hemorrhagica	...	1	...	...	...	1	1
	5	5	...	7	...	3	10
DISEASES OF THE MIND							
Dementia (paralytic)	1	...	...	...	1	...	1
Dementia (praecox)	...	1	...	...	1	...	1
Dementia (senile), pulmonary emphysema, arterio-sclerosis, chr. gastritis	1	...	...	...	1	...	1
Insanity, chr. nephritis	1	...	...	...	1	...	1
Mania (depressive)	...	1	...	...	1	...	1
	3	2	...	...	5	...	5
NERVES							
Neuralgia of facial nerves, anemia	...	1	...	1	...	...	1
Neuralgia of facial nerves, anemia, hemorrhoids	...	1	...	1	...	...	1
Neuralgia of intercostal nerves	...	1	1	...	...	...	1
Neuralgia of sciatic nerve	5	1	...	6	...	...	6
Neuralgia of sciatic nerve, chr. nephritis, cirrhosis of liver	1	...	1	...	...	...	1
Neuritis (brachial)	...	1	...	1	...	...	1
Neuritis (multiple)	...	1	...	1	...	...	1
Neuritis (peripheral)	1	3	...	3	1	...	4

	Male	F.	C.	Imp.	Un.	D.	Tot.
Nerves—Cont.							
Neuritis (peripheral), alcoholism	1	1	...	2	...	...	2
Neuritis (rheumatic)	...	1	...	1	...	...	1
Zoster	...	1	1	...	...	...	1
Zoster, arterio-sclerosis	...	1	...	1	...	...	1
	8	13	3	17	1	...	21
NERVOUS DISEASES OF UNKNOWN ORIGIN							
Catalepsy, hysteria	...	1	...	...	1	...	1
Chorea	...	1	1	...	...	...	1
Chorea, mitral insufficiency and stenosis	2	...	1	1	...	...	2
Chorea, patent ductus arteriosus	1	...	...	1	...	...	1
Exophthalmos (unilateral)	...	1	...	...	1	...	1
Epilepsy (petit mal), facial neuralgia	...	1	...	...	1	...	1
Hysteria	2	3	2	3	...	...	5
Migraine	2	1	1	2	...	...	3
Neurasthenia	4	14	...	17	1	...	18
Neurasthenia, refraction error	1	...	...	1	...	...	1
Neurosis (cardiac)	1	...	...	1	...	...	1
Neurosis (gastric)	...	2	...	2	...	...	2
Pseudo angina pectoris	...	1	...	1	...	...	1
Spasm (habit), hysteria	1	...	...	1	...	...	1
	14	25	5	30	4	...	39
SPINAL CORD							
Paralysis (general)	1	...	...	1	...	...	1
Paraplegia (ataxic)	2	...	...	1	1	...	2
Paraplegia (ataxic), Charcot's joint	1	...	...	...	1	...	1
Sclerosis (amyotrophic lateral)	...	1	...	...	1	...	1
Sclerosis (disseminated)	1	...	...	1	...	...	1
Sclerosis (lateral)	1	...	...	...	1	...	1
Sclerosis (multiple)	...	1	...	...	1	...	1
	6	2	...	3	5	...	8
OSSEOUS SYSTEM							
BONES							
Caries of spine, spastic hemiplegia, mitral stenosis and insufficiency	1	...	...	1	...	...	1
JOINTS							
Arthritis deformans	3	2	...	3	2	...	5
Arthritis deformans, hemiplegia, secondary anemia	...	1	...	...	...	1	1
Arthritis deformans, mitral insufficiency	...	1	...	1	...	...	1
Arthritis deformans (vertebral)	1	...	...	1	...	...	1
Chronic articular rheumatism, neurasthenia	...	1	...	1	...	...	1
Chronic articular rheumatism, nephritis	...	1	...	1	...	...	1
	4	6	...	7	2	1	10
REPRODUCTIVE SYSTEM							
OVARIES AND TUBES							
Cystic ovary	...	1	...	1	...	...	1
Salpingitis (acute)	...	2	...	1	1	...	2
	...	3	...	2	1	...	3
PREGNANCY, ETC.							
Pregnancy	...	1	...	...	1	...	1
Puerperal eclampsia	...	1	1	...	...	...	1
Vomiting of pregnancy	...	1	...	1	...	...	1
	...	3	1	1	1	...	3
UTERUS							
Abscess of broad ligament	...	1	...	1	...	...	1
Endometritis, salpingitis, neurasthenia	...	1	...	1	...	...	1
Retroversion of uterus, pelvic inflammation	...	1	...	...	1	...	1
	...	3	...	2	1	...	3

	Male	F.	C.	Im.	Un.	D.	Tot.
PROSTATE							
Hypertrophy of prostate	1	...	...	1	...	...	1
Prostatitis (chronic), chr. nephritis	1	...	...	...	...	1	1
Prostatitis (chronic), retention of urine, ischio-rectal abscess, nephritis, uremia	1	...	...	1	...	...	1
Prostatitis (acute), retention of urine, nephritis, uremia	1	...	...	...	...	1	1
	4	...	...	2	...	2	4
RESPIRATORY SYSTEM							
BRONCHI							
Asthma	...	3	...	3	...	...	3
Asthma, chr. nephritis	...	1	...	1	...	...	1
Asthma, bronchitis	...	4	1	3	...	...	4
Asthma, intestinal auto-intoxication	1	...	...	1	...	...	1
Bronchitis	1	9	5	5	...	...	10
Bronchitis (acute), carcinoma of breast	...	1	...	1	...	...	1
Bronchitis (acuate), epilepsy, hypertrophy of prostate	1	...	...	1	...	...	1
Bronchitis (acute), laryngitis, acute catarrhal otitis media	...	1	1	...	...	...	1
Bronchitis (acute), mitral insufficiency	1	...	...	1	...	...	1
Bronchitis (acute), myalgia	1	...	1	...	...	...	1
Bronchitis (acute), scarlet fever	...	1	...	1	...	...	1
Bronchitis (diffuse capillary)	...	1	1	...	...	...	1
	5	21	10	16	...	...	26
LARYNX							
Laryngitis	1	...	...	1	...	...	1
Laryngitis, tonsillitis	1	...	1	...	...	...	1
	2	...	1	1	...	...	2
LUNGS							
Abscess of lung	...	1	...	1	...	...	1
Emphysema (pulmonary), arterio-sclerosis, incontinence of urine	...	1	...	...	1	...	1
Emphysema (pul.), mitral insufficiency, chr. bronchitis	1	...	...	1	...	...	1
Emphysema (pul.) chr. bronchitis	1	1	1	1	...	...	2
Emphysema (pul.), bronchitis, arterio-sclerosis, myocardial insufficiency, erythematous eczema	1	...	...	1	...	...	1
Pneumonia (broncho—)	1	2	2	1	...	...	3
Pneumonia (broncho—), bronchitis, prolapse of uterus	...	1	...	1	...	...	1
Pneumonia (lobar)	29	14	19	2	...	22	43
Pneumonia (lobar, resolution delayed)	4	1	5	...	...	...	5
Pneumonia (lobar, unresolved)	...	1	1	...	...	...	1
Pneumonia (lobar), mitral stenosis	...	1	...	1	...	...	1
Pneumonia (lobar), dextra cardia	.	1	1	...	...	...	1
Pneumonia (lobar), oedema of lungs	...	2	...	...	...	2	2
Pneumonia (lobar), mitral insufficiency, aortic stenosis	...	1	...	...	...	1	1
Pneumonia (lobar), septic endocarditis	...	1	...	...	...	1	1
Pneumonia (lobar), cerebral embolism with hemiplegia	1	...	...	...	...	1	1
Pneumonia (lobar), chr. pulmonary tuberculosis	1	...	...	...	...	1	1
Pneumonia (lobar), acute otitis media	1	...	...	1	...	...	1
Pneumonia (lobar), pleurisy with effusion	2	...	2	...	...	...	2
Pneumonia (lobar), suppurative pleurisy	1	...	...	1	...	...	1
Pneumonia (lobar). phlebitis of leg	...	1	1	...	...	...	1
Pneumonia (lobar), chr. nephritis	...	1	...	...	...	1	1
Pneumonia (lobar), dry pleurisy	...	1	...	1	...	...	1
Pneumonia (lobar), parenchymatous nephritis	1	...	...	...	...	1	1
Pneumonia (lobar), multiple thrombosis, dry pleurisy, strongyloides intestinalis	1	...	...	1	...	...	1
Pneumonia (atypical)	2	...	2	...	...	...	2
Pneumonia (septic)	...	2	1	1	...	...	1
Pneumonia (septic, following abortion)	...	1	...	...	...	1	1
	47	34	35	14	1	31	81
PLEURA							
Pleurisy, secondary anemia, fibroid of uterus	...	1	...	1	...	...	1
Pleurisy (dry)	1	1	1	1	...	...	2
Pleurisy (dry), ac. nephritis	1	...	...	1	...	...	1
Pleurisy (fibrinous), ac. articular rheumatism	1	...	...	1	...	...	1
Pleurisy (suppurative)	1	...	...	...	1	...	1
Pleurisy convalescence	1	...	1	...	...	...	1
Pleurisy with effusion	7	2	5	3	1	...	9
Pleurisy with thickened pleura	...	1	...	1	...	...	1
	12	5	7	8	2	...	17

	Male	F.	C.	Imp.	Un.	D.	Tot.
SENSE ORGANS							
ORGAN OF HEARING							
Mastoiditis	...	1	...	1	...	...	1
Otitis media (catarrhal)	1	...	...	1	...	...	1
Otitis media (suppurative)	1	...	...	...	1	...	1
	2	1	...	2	1	...	3
ORGAN OF VISION							
Blepharitis, eczema	1	...	...	1	...	...	1
Detachment of retina	...	1	...	...	1	...	1
	1	1	...	1	1	...	2
TEGUMENTARY SYSTEM							
SKIN, ETC.							
Eczema	...	1	...	1	...	...	1
Necrosis of face following alcohol injections	...	1	...	1	...	...	1
Psoriasis, cystitis	1	...	...	1	...	...	1
Purpura hemorrhagica, intracranial hemorrhage	1	...	...	...	...	1	1
Purpura hemorrhagica, retinitis proliferans	...	1	...	1	...	...	1
Ulcer of leg	1	...	1	...	...	...	1
	3	3	1	4	...	1	6
URINARY SYSTEM							
KIDNEY							
Hematuria	1	...	...	...	1	...	1
Nephritis (acute)	...	1	...	1	...	...	1
Nephritis (chronic interstitial)	18	15	...	24	1	8	33
Nephritis (chr. in.), aortic insufficiency	2	...	...	1	...	1	2
Nephritis (chr. in.), abscess of lung	1	...	...	...	...	1	1
Nephritis (chr. in.), aortic regurgitation	1	...	...	...	...	1	1
Nephritis (chr. in.), appendicitis, arterio-sclerosis	1	...	...	1	...	...	1
Nephritis (chr. in.), arterio-sclerosis	4	...	...	4	...	...	4
Nephritis (chr. in.), ascites, hydrothorax	...	1	...	1	...	...	1
Nephritis (chr. in.), anemia, arterio-sclerosis	1	...	...	1	...	...	1
Nephritis (chr. in.), bronchitis, emphysema	...	1	...	1	...	...	1
Nephritis (chr. in.), cardio-fibrosis	1	...	...	1	...	...	1
Nephritis (chr. in.), constipation	...	1	...	1	...	...	1
Nephritis (chr. in.), debility	...	1	...	1	...	...	1
Nephritis (chr. in.), dilatation of heart, anemia, arterio-sclerosis	1	...	...	...	...	1	1
Nephritis (chr. in.), dry pleurisy, pericarditis	1	...	...	1	...	...	1
Nephritis (chr. in.), hydrothorax	1	...	...	1	...	...	1
Nephritis (chr. in.), mitral stenosis	1	...	...	...	1	...	1
Nephritis (chr. in.), myocardial insufficiency	1	...	...	1	...	...	1
Nephritis (chr. in.), myocarditis	...	1	...	1	...	...	1
Nephritis (chr. in.), myocarditis, mitral and aortic insufficiency	...	1	...	1	...	...	1
Nephritis (chr. in.), oedema of lungs	2	1	...	1	...	2	3
Nephritis (chr. in.), pericarditis, mitral stenosis and insufficiency	1	...	...	...	...	1	1
Nephritis (chr. in.), uremia	1	5	...	2	...	4	6
Nephritis (chr. in.), uremia, hemiplegia	1	...	...	...	...	1	1
Nephritis (chr. in.), uremia, mitral and aortic insufficiency	...	2	...	...	...	2	2
Nephritis (chr. in.), uremia, myocarditis	...	1	...	1	...	...	1
Nephritis (chr. in.), varicose veins, ac. thrombo-phlebitis	...	1	...	1	...	...	1
Nephritis (chr. in. with ac. exacerbation)	3	...	...	3	...	...	3
Nephritis (chr. in. with ac. exacerbation), pulmonary tuberculosis	1	...	...	1	...	...	1
Nephritis (chr. in. with ac. exacerbation), ulcerative colitis, prostatitis	1	...	...	1	...	...	1
Nephritis (chr. parenchymatous)	1	...	...	...	1	...	1
Nephritis (sub-acute), epilepsy	...	1	...	1	...	...	1
Pyelitis	3	6	5	4	...	...	9
Pyonephrosis	1	...	...	...	1	...	1
	50	39	5	57	5	22	89
URINARY BLADDER							
Cystitis	1	4	3	2	...	...	5
Cystitis, hypertrophy of prostate	1	...	...	1	...	...	1
Retention of urine	1	...	...	...	1	...	1
	3	4	3	3	1	...	7

	Male	F.	C.	Imp.	Un.	D.	Tot.
DISEASES DUE TO ANIMAL PARASITES							
Scabies, eosinophilia	1	...	1	...	...	...	1
Strongyloides intestinalis, catarrhal cholangitis	1	...	...	1	...	...	1
Trichiniasis	...	2	1	1	...	...	2
	2	2	2	2	...	...	4
GENERAL INJURIES							
Heat prostration	...	1	1	...	...	...	1
Strangulation, shock, nervous exhaustion	...	1	1	...	...	...	1
	...	2	2	...	...	...	2
LOCAL INJURIES							
Ammonia burns of mouth and throat	...	1	...	1	...	...	1
Carbolic acid burn of arm	...	1	1	...	...	...	1
Fracture of rib	...	1	...	1	...	...	1
Fracture (old) of femur	...	1	...	1	...	...	1
Laceration of perineum	...	1	...	...	1	...	1
	...	5	1	3	1	...	5
NEOPLASMS							
Carcinoma of breast, mediastinal metastases	...	1	...	...	...	1	1
Carcinoma of breast and axillary nodes, secondary neuritis	...	1	...	...	1	...	1
Carcinoma of colon	...	1	...	...	...	1	1
Carcinoma of liver	2	...	...	...	...	2	2
Carcinoma of lung, metastases in larynx, and in cervical, axillary and mesenteric nodes	1	...	...	...	...	1	1
Carcinoma of mediastinum	...	1	...	...	...	1	1
Carcinoma of oesophagus	1	...	...	1	...	...	1
Carcinoma of ovary	...	1	...	...	1	...	1
Carcinoma of pancreas	...	1	...	1	...	...	1
Carcinoma of pylorus	...	1	...	...	1	...	1
Carcinoma of sigmoid	...	1	...	1	...	1	...
Carcinoma of stomach	4	6	...	2	6	2	10
Carcinoma of stomach, cirrhosis of liver, secondary anemia	1	...	...	1	...	...	1
Carcinoma of stomach, metastases in liver and forehead	...	1	...	...	1	...	1
Hypernephroma, coccygodynia	...	1	...	...	1	...	1
Osteo-sarcoma of sacrum	1	...	...	...	...	1	1
Sarcomatosis (general)	1	...	...	...	...	1	1
Tumor of brain	1	1	...	1	1	...	2
Tumor of brain, pulmonary tuberculosis	1	...	...	...	1	...	1
Tumor of mediastinum	2	...	...	1	1	...	2
Tumor of pelvic region	1	1	...	1	1	...	2
Tumor of sacro-iliac region	1	...	...	...	...	1	1
	17	18	...	9	15	11	35
INTOXICATIONS AND POISONS							
EXOGENOUS INTOXICATIONS							
Alcoholism (acute)	4	1	3	2	...	...	5
Alcoholism (acute), cirrhosis of liver, entero-colitis, gastritis, multiple neuritis, delirium tremens	...	1	...	1	...	...	1
Alcoholism (acute), hemorrhoids	1	...	...	1	...	...	1
Alcoholism (acute), sinus of inferior maxilla	1	...	1	...	...	...	1
Alcoholism (chronic), cirrhosis of liver	1	1	...	...	2	...	2
Alcoholism (chr.), cirrhosis of liver, pulmonary tuberculosis	1	...	...	2	...	...	1
Alcoholism (chr.), Korsikoff's syndrome	...	1	...	...	1	...	1
Alcoholism (chr.), neurasthenia, insomnia	1	...	...	1	...	...	1
Cocainism, morphinism, constipation	1	...	...	1	...	...	1
Morphinism	...	4	2	1	1	...	4
Morphinism—heroin	1	...	...	1	...	...	1
Chronic poisoning by chloral, neurasthenia	1	...	...	1	...	...	1
Chronic poisoning by lead	1	...	1	...	...	...	1
Chronic poisoning by mercury	...	2	...	2	...	...	2
Chronic poisoning by sulphonal, neurasthenia	...	1	...	1	...	...	1
Acute poisoning by ptomaine	...	1	1	...	...	...	1
	13	12	8	13	4	...	25
ENDOGENOUS INTOXICATIONS							
Auto-intoxication, enteritis	2	...	2	...	...	...	2
Auto-intoxication, nephritis	...	1	...	1	...	...	1
Diabetes mellitus	3	5	...	6	...	2	8
Diabetes mellitus, hypertrophy of prostate, pulmonary tuberculosis	1	...	...	1	...	...	1

	Male	F.	C.	Imp.	Un.	D.	Tot.
Endogenous Intoxications—Cont.							
Diabetes mellitus, hysteria	...	1	...	1	...	...	1
Diabetes mellitus, lobar pneumonia	1	...	...	...	...	1	1
Diabetes mellitus, nephritis	...	3	...	2	1	...	3
Diabetes mellitus, pulmonary tuberculosis	...	1	...	1	...	...	1
Gout	2	...	1	1	...	...	2
Gout, diabetes mellitus, nephritis	1	...	...	1	...	...	1
	10	11	3	14	1	3	21
ANIMAL POISONS							
Acute poisoning by fish	...	1	1	...	...	...	1
MISCELLANEOUS CONDITIONS							
Debility	1	1	...	2	...	...	2
Unclassified fever	2	2	3	...	1	...	4
	3	3	3	2	1	...	6

SUMMARY

	Male	F.	C.	Imp.	Un.	D.	Tot.
Micro-organic Diseases	171	208	174	142	17	46	379
Alimentary System	70	73	58	69	13	3	143
Cardio-vascular System	48	54	2	53	11	36	102
Connective Tissue	2	1	....	2	....	1	3
Ductless Glands	1	3	....	1	1	1	4
Muscular System	2	....	....	1	1	....	2
Nervous System	36	47	8	57	15	3	83
Osseous System	5	6	....	8	2	1	11
Reproductive System	4	9	1	7	3	2	13
Respiratory System	66	60	53	39	3	31	126
Sense Organs	3	2	....	3	2	....	5
Tegumentary System	3	3	1	4	....	1	6
Urinary System	53	43	9	60	6	22	96
Animal Parasites	2	2	2	2	....	....	4
General Injuries	....	2	2	....	....	....	2
Local Injuries	....	5	1	3	1	....	5
Neoplasms	17	18	....	9	15	11	35
Intoxications	23	24	12	27	5	3	47
Miscellaneous	3	3	3	2	1	....	6
	509	563	325	489	96	162	1062

REPORT OF THE TREATMENT OF TYPHOID FEVER WITH VACCINE.*

Austin W. Hollis, M.D.

During my service at St. Luke's Hospital in the late summer and fall of 1909, I began the treatment of all cases of typhoid fever with vaccine. A review of the literature at that time showed the treatment to be still in the experimental stage, with doses ranging from 1,000,000 to 300,000,000 dead typhoid bacilli, and no uniformity as to the repetition of the dose. In our cases the dose was given subcutaneously in the region of the buttocks every second or third day, each dose being either 10,000,000 or 50,000,000 dead bacilli. During the summer and fall we had 37 cases of typhoid fever; some of these cases occurred in the service of Dr. S. W. Lambert, and, being of the same season, are introduced for comparison.

Twenty-one (21) cases were treated by the older method of hydrotherapy, either by the tub bath or sponging.

Eleven (11) cases were treated with vaccine without hydrotherapy.

The remaining five (5) cases were too short a time under treatment to make any comparisons.

The diet in general, especially in the hydrotherapy cases, was of the high caloric variety. No drugs were employed, but strychnine and whiskey were used as stimulants in the severe cases. The bowels were moved by enemata.

The cases which were treated by hydrotherapy, barring a few which were given sponge baths because of poor response to tubbing, had a tub bath from 80° to 90° for 15 minutes with friction every four hours from noon until midnight inclusive if the rectal temperature was 103° or higher. At 8 a.m. all received a warm soap and water sponge. Those treated by vaccination received no baths other than a daily warm soap and water sponge. In a very few cases 250,000,000 bacilli were employed but no different clinical or laboratory effect could be noticed between the smaller and larger doses. No fever reaction was obtained. No case ended by crisis. Occasionally a slight local erythema or itching was noticed at the point of injection, which cleared up without treatment. The agglutinins were markedly stimulated by the vaccines. Complete agglutination was frequently observed in dilutions as high as 1-500 to 1-1000, while cases without vaccines showed incomplete agglutination in 1-80 dilution. The febrile course in a few cases seemed shortened, but the abrupt change from the sustained fever to the amphobolic type seemed hastened by vaccination, averaging 17.1 in the cases treated by vaccines, and 23.2 in those getting tub baths.

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The average day of admission from the beginning of the first symptom was 11.3 and 13.8; of defervescence 36.7 and 34.3 for the vaccination and hydrotherapy cases respectively. The temperature averaged equally high in both series. Hemorrhage was seen with equal frequency, though not so severe, in the vaccinated cases. Relapses were noted with more frequency in the vaccinated cases—30 per cent as compared with 10 per cent. Relapses were in all cases mild. The cases which received vaccine were in no way selected.

No death occurred in the vaccinated cases; four occurred in the other series, one from hemorrhage, one from toxemia, one from perforation and one from pulmonary thrombosis with necrosis of the whole left upper lobe of the lung.

Headache, gastrointestinal symptoms and toxemia were far less frequent in the vaccinated series than in the other cases, and the absence of discomfort seemed striking, while convalescence was more rapid.

A comparison of possible value of the effects of vaccine may be made in three cases which occurred in young adults of the same family in the better class of life who apparently were infected by oysters at about the same time.

The first, a robust man of 26 years, entered the hospital about five days before the other two. He received tub baths throughout his illness; he was profoundly sick; his temperature held at 104° to 105° for 39 days before assuming the amphobolic type; the latter stage of the disease was associated with severe chills and violent temperature reactions, once or twice reaching 107°, which probably came from a cholecystitis; he defervesced on the 62d and made an uninterrupted recovery.

The other two cases, husband and wife, entered the hospital together. The man, 33 years, also robust, had a temperature of 104° and marked toxemia; on the sixth day of his disease he received 50,000,000 dead bacilli injected subcutaneously; the dose was repeated every other day until the 14th. The response was rapid; by the eighth day, still with temperature of 104°, he was bright mentally and clamoring for food and said he felt like getting up. On the 13th day the fever broke and on the 17th day was normal. A very large hard spleen did not diminish in size, and on the 20th day fever returned, and he had a nine-day relapse of very mild type with fever as the only symptom. Vaccination was resumed on the 20th day, 50,000,000 dead bacilli being given every other day until the 32d. Convalescence was very rapid and complete.

The third case, the woman, presented three serious complications. She was 4 months pregnant, had a history of a former dilatation of the heart and carried a weak pulse ranging from 120 to 150 throughout the course of the disease. She had repeated profuse nasal hemorrhages which were controlled with great difficulty. For a few days she was treated by sponge baths, but her reaction was poor and because of exhaustion they had to be stopped. Vaccine was then employed reluctantly, because personal experience was at that time very limited; but on the 10th day we started with 10,000,000 dead bacilli every other day and later raised the dose to 50,000,000. The temperature continued to hold at 104° to 105° for twenty-one days; she was exceedingly weak, but her apathy and toxemia disappeared very shortly after vaccination was begun. She was fearful of the epistaxis and the dangers of abortion. The fever was prolonged to the 47th day, but after the 21st it became quite moderate and she appeared in a convalescent stage. During February of this year she gave birth to a healthy boy.

A fourth case occurred in the epidemic above 116th street. I saw her first in consultation with Dr. Almgren. She was stuporous, moaned from severe occipital headache, and the diagnosis was in doubt. She was taken to St. Luke's on the sixth day of her illness and given sponge baths, with icebags to head. It was not before the ninth day that positive blood reaction was manifested. On the tenth day 10,000,000 bacilli were injected and continued every other day. Response was very rapid, with marked amelioration of the headache, and nourishment was taken better. She rested quietly. In spite of the sponge baths her fever until the 10th day held very closely to 104° or a little over; but after the administration of the vaccine it never went above 104° and soon entered the amphobolic stage. Defervesence occurred on the 24th day. Convalescence was rapid, but one day there was a temperature of 101°, due to an abortive otitis media.

Conclusions.—It would be absurd to assume any positive value from vaccine therapy in typhoid fever in such a small series of cases; it would be equally absurd to rely on the impressions even of an old observer as to the benefit of such treatment on the same series. When we hear reports that starvation diet and the high caloric diet are both highly successful in typhoid fever; when after years of experience one has adopted what seems to be a rational diet and returns to the hospital service to hear the house physician tell of the wonderful results of the "Coleman" or "Shattuck" feeding; when we read of the annual fights of the pediatrists over the high proteid and high carbohydrate diet in infant feeding we realize the difficulties of observation without prejudice. But when the answer is plainly written, as it was in the case of diphtheria antitoxin, although there may have been bitter differences of opinion in the beginning, yet in time those who got the answer first were vindicated by the truth of their observations.

In our experience in the vaccine therapy of typhoid fever we can claim that the agglutinins were markedly stimulated, and there were no harmful results.

Are there any theories that may be advanced to capture the intelligence in this treatment?

Cure in all disease due to bacteria is probably accomplished by the autoprotective forces of the blood. As far as our present knowledge goes, these diseases are all local infections; they may become general from multiple foci; bacteriemia may result but the blood stream itself is not the culture medium. Bacteria may be found in it, but never easily and usually by a difficult cultural process. In spite of the fact that of late years typhoid fever has often been spoken of as a general blood infection, there is no evidence to show that Peyer's patches and the spleen are not the early sites of the bacterial growth, with other foci only as complications. In no sense may it be considered a blood infection simply because the blood may hold bacteria just as the urine may.

Fever is due to some specific reaction between living cells or their plasma and bacteria or other living organisms or their products. It is not due to dead bacteria or their products in artificial culture media. An effort has been made to prove fever a beneficial and necessary protective reaction; but that it is ever beneficial is extremely doubtful. The employment of antitoxin serum shows that fever is not an essential reaction in its curative action. The familiar vaccination in smallpox produces full protection by a local reaction, and not because of a febrile movement. The argument that it would seem useless, even harmful, to introduce into the system the bodies of dead bacteria to cure a disease which is constantly poisoning the host with living bacteria or their products is a very superficial one. We assume that the cure in bacterial diseases is by the autoprotective forces of the blood, stimulated by the bacteria of the same disease or their products.

In pneumonia the onset is usually with a chill from toxic absorption; a vast capillary surface is exposed, and the clinical signs are those of a violent toxemia; if the protective forces respond, they do so quickly and decisively. In typhoid fever action is similar but slower. In these diseases we do not know how many bacteria or how much toxin is being absorbed by the minute; we do know that up to a certain point the system can stand the harmful effects and that it is protecting itself. If we can artificially stimulate the agglutinins, the opsonins, the precipitins and antibodies by the injection of dead bacteria which do not increase the fever nor add to the patient's danger, then we can say that the use of vaccine rests upon a sound basis; but to get the best results a great deal more is necessary to know; we do not know how often it should be employed, whether the dose should be uniform, whether it should begin very early with a large initial dose, or with small and increasing doses. The argument that we should use much larger doses because antitoxin is now employed in much larger doses than in the beginning is altogether fallacious. That we use much smaller doses of tuberculin now than formerly may be more reasonably argued. It has been conclusively shown that billions of dead typhoid bacilli may be introduced into the well without harmful reaction; we do not know certainly whether the same holds good in the case of those suffering from the disease.

From theory alone I predict a brilliant future for typhoid vaccine as a protective as well as a therapeutic agent.

To Dr. William R. Janeway, my house physician, who prepared the statistics of this report and aided me in the treatment of the cases, I give my sincere thanks.

SOME RECENT EXPERIENCES WITH THE NEW SPECIFIC REMEDY—EHRLICH 606

SAMUEL W. LAMBERT, M.D.

The presentation of a report on the new remedy of Ehrlich known as 606 based only on a short clinical experience can be justified in part by the rapidity of action of the drug but more particularly by the great interest which has been roused in the medical world by this new conception of its discoverer. Ehrlich has added another brilliant achievement to his list of successes. Not only has he formulated a new idea in therapeutics christened by him a "therapia sterilisans magna" by which he proposes with a single dose to destroy the germs of a disease that untreated would outlast the life of the individual and be transmitted to his descendants. But also Ehrlich has given us the remedy which tries to accomplish this seeming impossibility as viewed by former medical standards.

Never before had such numbers of reports from various medical sources been so unanimous in support of the claims for a new therapeutic measure. The future may show that the claims for the method and the drug are exaggerated but enough is known to prove the principal facts that the large majority of syphilitic patients may be freed of all visible signs of the disease by the injection of a single dose of Ehrlich 606. In this paper it is proposed to review the question from a practical standpoint and to report a few typical cases.

One of the most interesting features of the subject is to be found in a study of the theoretical consideration by which Ehrlich reached his conclusions of the possibility of a single curative dosage. He began his work in this line of study on trypanosomes and sought for remedies which would be parasiticide on the one hand and on the other would not destroy the tissue cells of the host. Ehrlich has invented two new terms to express these ideas "parasito tropic" and "organo tropic."

The group of parasites to which the spirochaetes belong includes the malarial plasmodia as well as the trypanosomes. Attention had been called to the use of arsenic as atoxyl in the trypanosomiasis of sleeping sickness and from this starting point Ehrlich began to experiment with chemical substitution products.

He found also that this group of animal parasites would become immune to arsenic compounds when the drug was given in divided doses, he therefore sought for a drug which would be so strongly parasitotropic and so weakly organotropic that in a single dose it would kill all the disease germs present in the body of the patient while sparing the many tissue cells of the host.

His first success was with trypanosomes in mice and with a new remedy named "trypan-red" with which he first successfully sterilized with a single dose the living mouse to trypanosome infection. He then turned his attention to other members of this group of parasites notably the spirochaetes and to the treatment of syphilis.

Ehrlich has experimented with more than 630 remedies and No. 606 on this list has seemed to fulfil the condition most accurately.

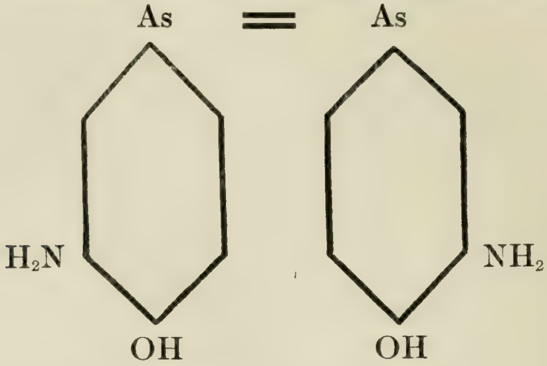
The testing on animals having proved its usefulness it was given to man and the new therapeutic idea of a therapia sterilisans magna was made public.

This acid salt is a yellow powder which oxidizes on exposure to the air and it must be preserved in vacuum tubes. This acid salt must be changed to the alkaline sodium salt or to the insoluble base suspended in a neutral mixture before it is injected or its administration will be very painful or even devoid of result. In one case known to the writer the preparation was faulty and a large swelling resulted at the seat of injection. After some days this was incised and a quantity of the remedy was found in a cavity with necrotic walls.

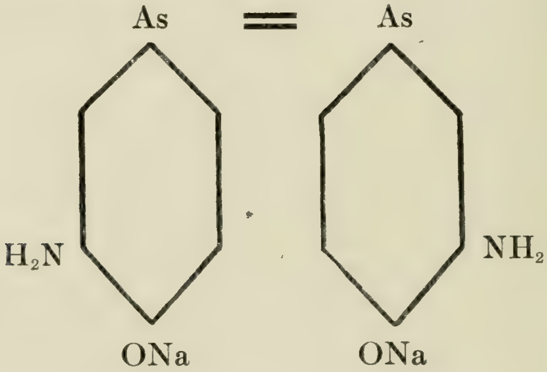
The preparation of the individual dose should be done immediately before injecting it either as the base itself after the method of Wechselmann or as the alkaline salt in the manner of Lesser. In both methods sterile utensils must be used and the strictest asepsis observed.

The most efficient method seems to be that of Lesser and consists of the preparation of a solution of the alkaline salt in as nearly neutral a solution as possible. The detail of the Lesser method is as follows: Dissolve the 606 salt in 15 cc. of hot water in a sterile glass-stoppered and graduated cylinder, then add 2 cc. of normal solution of sodium hydrate. At first a precipitation occurs which disappears upon the addition of a sufficient quantity of the sodium hydrate. This should be added drop by drop until a clear solution is obtained which should be neutral in reaction. This is diluted with sterile water to 20 cc. and injected, preferably in the buttocks.

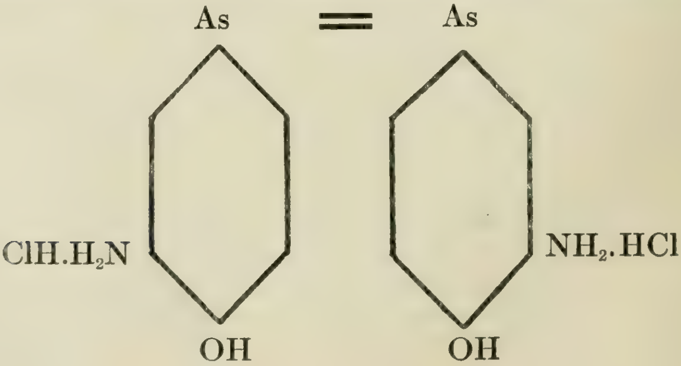
This remedy is a preparation of arsenic to be known chemically as dioxydiamidoarsenobenzole—its chemical formula is $C_{12} H_{12} O_2 N_2 As_2$ arranged graphically thus:



Its alkaline salt with sodium is graphically this:



It is distributed in the form of an acid salt, a dihydrochloride shown graphically thus:



The Wechselmann method consists of neutralizing the acid salt with sodium hydrate and then suspending the resulting base in sterile water which is neutral in reaction.

The dosage is from 0.35 grams to 0.6 grams for adults and proportionately reduced for children. A recent publication by Duhot of Brussels gives 1.0 gram as the regular dose for a strong otherwise healthy man. The dose for nursing children should be at least one-twelfth the smallest adult dose; it is to be estimated at 0.008 grams per kilo gram of weight. The nursing child can be cured of its hereditary syphilis through the mother's milk after the mother has received an injection, but in severe cases, and in bottle fed children an injection must be done.

The injections have been made intravenously, intramuscularly in the gluteal region and subcutaneously in the inter scapular region. In the first four cases quoted below the injections were given subcutaneously and the drug was reduced to the neutral base, in the fifth case the 606 was injected as the alkaline salt.

The known power of the original arsenical parasiticide of this series—atoxyl—to cause an atrophy of the optic nerve has led to a certain suspicion against all arsenic-containing drugs and Ehrlich 606 has been carefully studied to discover such an effect if present. No such cases have been reported in the literature but Ehrlich has formulated certain precautions to be observed in the use of the drug both because of this tendency to cause optic nerve disease of other preparations of arsenic and because the drug has hastened death in persons suffering from chronic disease of the heart, the kidneys and the nervous centres. These indications for care and caution in its use are the following: Signs of disease of the optic nerves, advanced disease of the heart or kidneys; tuberculosis; old age or cachexia from any cause and disease of the brain even if that be of syphilitic nature. It is to be expected that such a powerful remedy should show some manifestations during its use. These therapeutic symptoms are pain which is extreme if the acid salt is used and may require morphine for its control. There is a local reaction with swelling and evidences of an inflammation but an abscess should not result if the injection is aseptically done and the drug is properly prepared. An eruption may follow which may be either a local erythema or a general urticaria and sometimes a reddening of the syphilitic rashes may result.

General symptoms of fever even to 104° of headache and gastric disturbance may last for several days. The patient is often constipated and may have difficulty in urinating during the same period. Two further precautions should be noted. The patient should be in bed under observation during this period of reaction and the diagnosis of syphilis should be established with every aid from the Wassermann test, and the examination for spirochaetes. The results of treating syphilis with Ehrlich's Arseno-benzole have been rapid and wonderfully uniform. All observers unite in the opinion that the method and the remedy show a specific action against syphilis which is remarkable.

The drug has been in use just over one year and recurrences and incomplete cures are beginning to be reported but nevertheless the enthusiasm of the medical men reporting such semi failures is still unbounded.

Under this treatment the spirochaetes disappear from the lesions both primary and secondary in from 24 hours to 7 days and the Wassermann reaction changed from positive to negative in from 12 days to 7 weeks. The action on primary chancres seems to vary with the age of the infection. An early infection is more difficult to influence than a chancre which has run its first period of induration.

It has been suggested that such chancres with extensive induration be excised as a help in the cure. When not excised a second dose has been found necessary. The secondary lesions have usually yielded quite promptly to a single dose. The tertiary lesions also have been quickly influenced for cure by 606 according to the reports of all observers. The future course of the disease cannot be formulated at present for no "cure" under the old treatment with mercury and iodide could ever be considered as a cure until at least two years had elapsed after the cessation of treatment. Arseno-benzole has been in use only 14 months and it has not been possible to apply this test of time which cannot be omitted from the study of these cases if a true conclusion is to be reached.

The following cases present many points of interest in this treatment:

Case I.—Hereditary Syphilis; female child 2 months old. Parents have been married one year, this is the first and only child, baby has been breast fed. At birth baby had a severe coryza. At seven weeks of age a rash came on the feet and hands which has spread gradually over the extremities, the face and the chest. The rash does not itch. Physical examination shows a fairly well nourished child covered with a dull red rash occurring in irregular confluent patches which are slightly raised. The

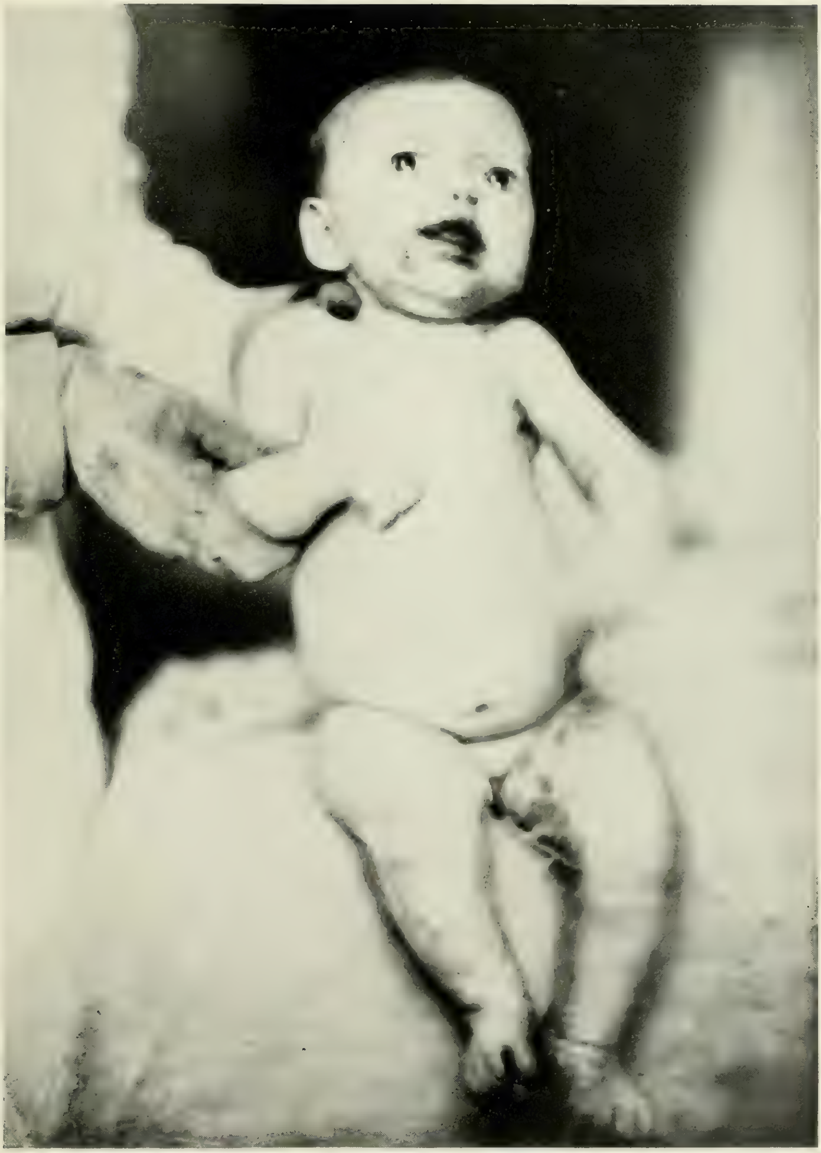


Fig. 1.—Case I. Hereditary Syphilis.

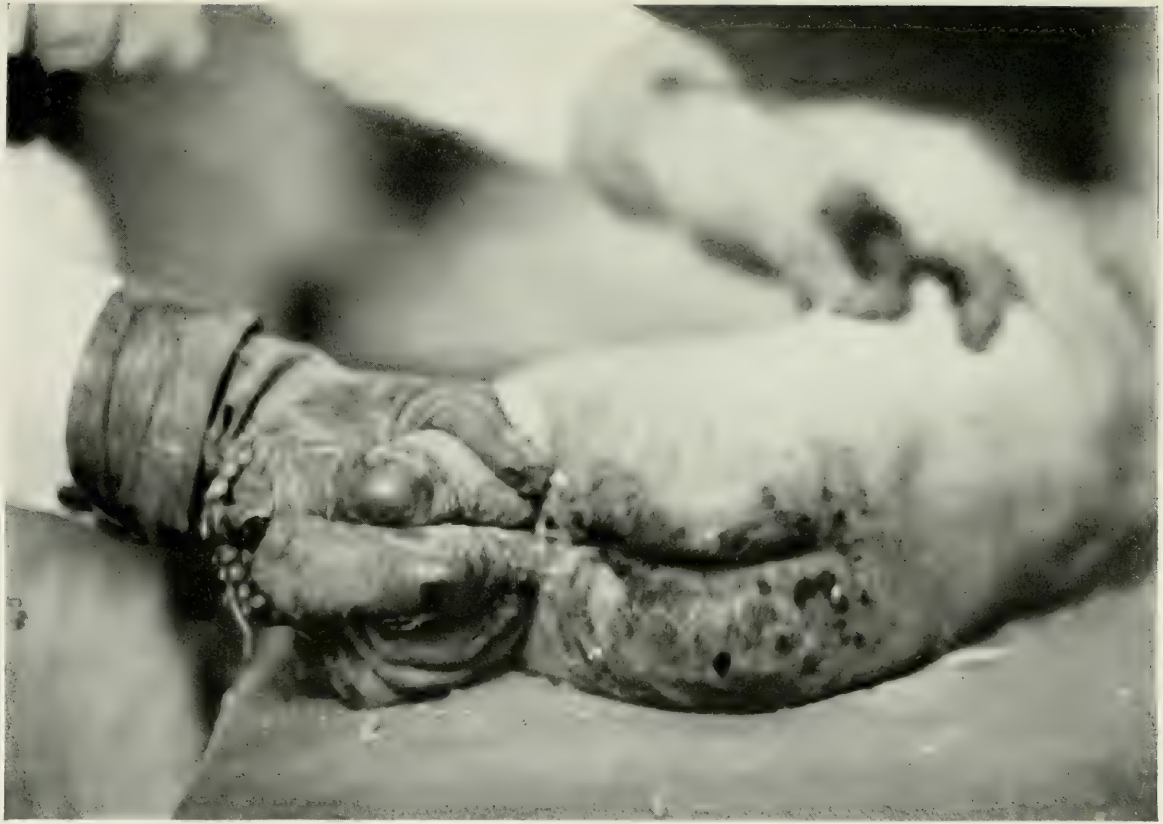


Fig. 2.—Case I. Hereditary Syphilis.

rash is general except the abdomen and is not plentiful on the chest and back. There are bullae on the thighs and the palms and soles are desquamating in large flakes. The mucous membranes are free except for labial fissures, there is a typical mucous patch on the left lip. The liver is enlarged two fingers below the costal margin and the spleen projects four fingers below the ribs. There is tenderness at upper epiphysis of humerus. Lymphatic glands are not enlarged. The anterior fontanelle is open, the eyes and ears are negative to examination. The nose is swollen and there is a nasal discharge. The baby's cry is hoarse, the heart and lungs seem normal.

The child had a fever from 100 to 102 for two days. A white blood cell count showed 32,000 leucocytes with 7% polymorphonuclears and 93% lymphocytes with normoblasts and megaloblasts. The Wassermann blood reaction was positive but spirochaetes were not demonstrated. On the third day of observation 25 milligrams of the arseno-benzole were injected under the skin of the back. No special reaction followed except local tenderness. Patient began to improve at once in all particulars, the hoarse cry lessened and the skin became less angry looking; ten days after injection the baby is much improved in the appearance of the face, the fissured mouth is almost healed, the liver and spleen are smaller. The white blood cell count is 20,000 with polymorphoneutrophiles 33%, lymphocytes 65%, eosinophiles 2%. Three weeks after the injection the baby appears with normal skin and mucous membranes and no coryza, with no enlargement of the liver or spleen. Her blood shows still a Wassermann reaction. The blood of both the father and mother were negative to the Wassermann reaction. The father admits having contracted syphilis 8 years previously for which he was treated one year and a half.

Case II.—Primary Syphilis. A man 32 years old. Thirty-one days before admission he noticed a small red sore on each lip, several days later a swelling came under the right angle of his jaw, he has had no rash but has suffered from headaches, poor appetite and pain in lip at night. Thinks he had a chancre 9 years ago with only a short period of treatment, he has had small-pox and measles.

Physical examination is negative except for two ulcers, one about 1 c. m. in diameter is to the right of the center on the upper lip and one about 1½ c. m. in diameter is on the left side of the lower lip, both are on an indurated hard base and covered with greyish sloughs. Both lips are greatly thickened and bulge out with the induration. Two large glands and many small ones are felt in the neck, submaxillary region and especially on the right side. There is no skin eruption. Smears from the ulcers show spirochetes and the Wassermann test is positive. The white blood count is 13,700 with polymorphonuclear 63%, lymphocytes 32%, eosinophiles 5%. The urine shows a faint trace of albumen. The eye grounds are normal. An injection of 0.6 gram of arseno-benzole was made on admission. There was a local reaction with pain and redness and a febrile action up to 101.8° for 25 hours.

Six days later the patient was discharged with slight improvement in the local condition if any. At this time spirochaetes were demonstrated again in the lesion. He was readmitted 14 days later, the condition of the chancres on the lips was practically unchanged, the glandular swellings under the jaw were slightly smaller. There was an eruption covering the entire chest, abdomen and all but the lowest part of the back. This rash is a typical dull red maculo papular secondary syphilid.

The Wassermann test was still positive and spirochaetes were found in the smears from the labial ulcers. The white blood cells count was 8,000 with a differential of polymorphonuclears 66%, lymphocytes 29%, eosinophiles 4%, basophiles 1%. A second injection of 0.6 gram of 606 was followed by a slight febrile reaction to 100.2° for 24 hours. Improvement has been slow—two weeks after the injection the rash has faded materially but smears from the lip do not show spirochaetes. The induration about the chancres has diminished. There has been no marked change for the better although a slow improvement is going on.

Case III.—Primary Syphilis. A woman, aged 27. Six weeks ago she noticed a small sore on the lower lip which was red and about ⅜ of an inch in diameter. This sore has persisted and gradually grown larger and has exuded a thin purulent fluid. Ten days after onset the glands under the jaw began to swell and be painful and she soon developed headaches which continue. Five days before admission she first noticed an eruption on the abdomen.

Physical examination showed all organs negative except a palpable spleen. There was a small indurated ulceration on the left side of the lower lip, the submaxillary lymph nodes are enlarged on the left side, no lesions in other glands or in the mouth. There was a maculo papular syphilid over the whole trunk of the body.

The Wassermann reaction was positive but spirochaetes could not be demonstrated in the ulcer on lip. The white blood cell count was 15,000 with a differential of polymorphonuclears 76%, lymphocytes 24%. An injection of 0.5 gram of 606 was followed by local pain for 48 hours and a febrile action to 100.8° for 24 hours.

Improvement has been rapid. The rash had disappeared at the end of four days and the primary lesion was distinctly smaller and softer after three days. This improvement has continued up to the present time.

Case IV.—Tertiary Syphilis; Dactylitis; Orchitis. A man, age 27. He had chancre six years ago followed by rash and sore throat in three weeks. Since then he has been under continuous medical advice and has taken mercurials as directed in an intermittent manner both by mouth and by inunction until four months ago.

Three months ago, he noticed a swelling of the index finger of the right hand which persists in spite of an incision which is now healed. During past three years there has been an intermittent swelling of the right testicle which is now extreme.

Patient took an atoxyl cure for a month, four months ago, with no result and has had no treatment for three months.

Physical examination shows no gross lesions of the organs, there are some pigmented scars on the legs. The first inter-phalangeal joint of the index finger of the right hand is twice its normal size; the swelling tapers off towards the tip; the swelling is hard and moderately tender and the other joints of the finger have a limited movement.

The right testicle is swollen, hard and tender and there is a hydrocele in addition. The blood shows a positive Wassermann reaction, the white blood cell count is 12,600 and the differential is polymorphonuclears 77%, lymphocytes 22%, eosinophiles 1%. An injection of 0.5 gram arseno-benzole caused a marked local reaction with a febrile rise to 100.2°.

The patient was convinced twelve days later that both lesions were improved. The dactylitis seemed distinctly better with less swelling and tenderness. The orchitis has not been affected.

Case V.—Tertiary Syphilis. A man, age 30. Patient contracted syphilis March 24, 1910, his chancre developed with bilateral inguinal buboes and was followed in a

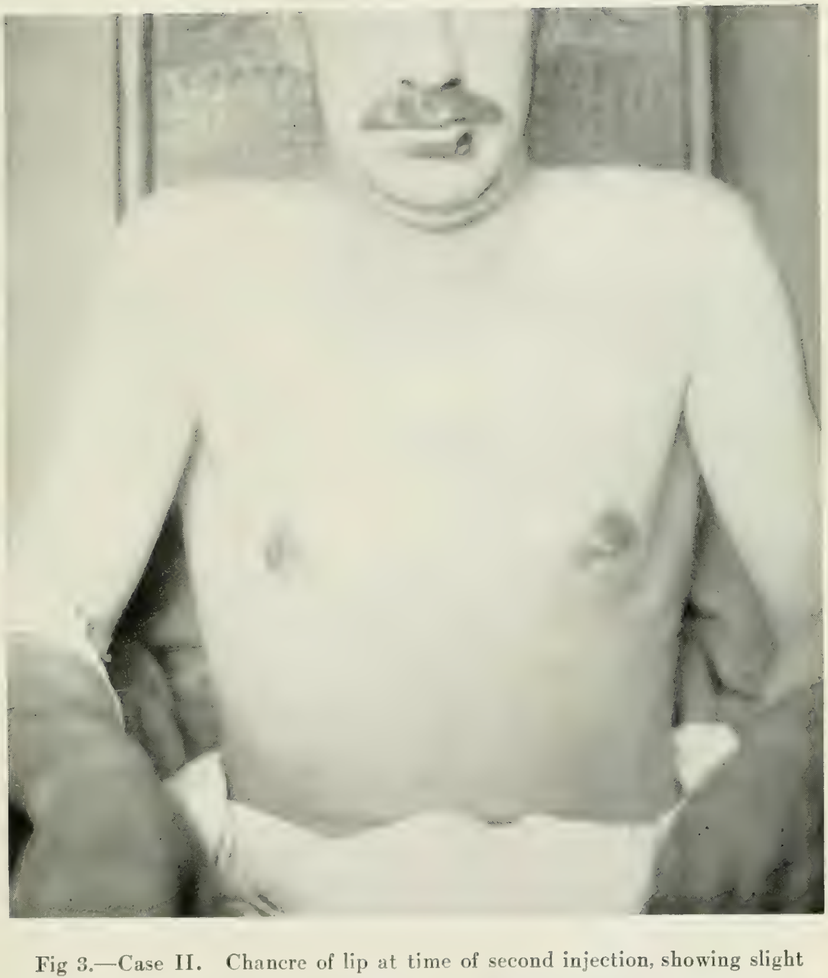


Fig 3.—Case II. Chancre of lip at time of second injection, showing slight improvement from first injection.

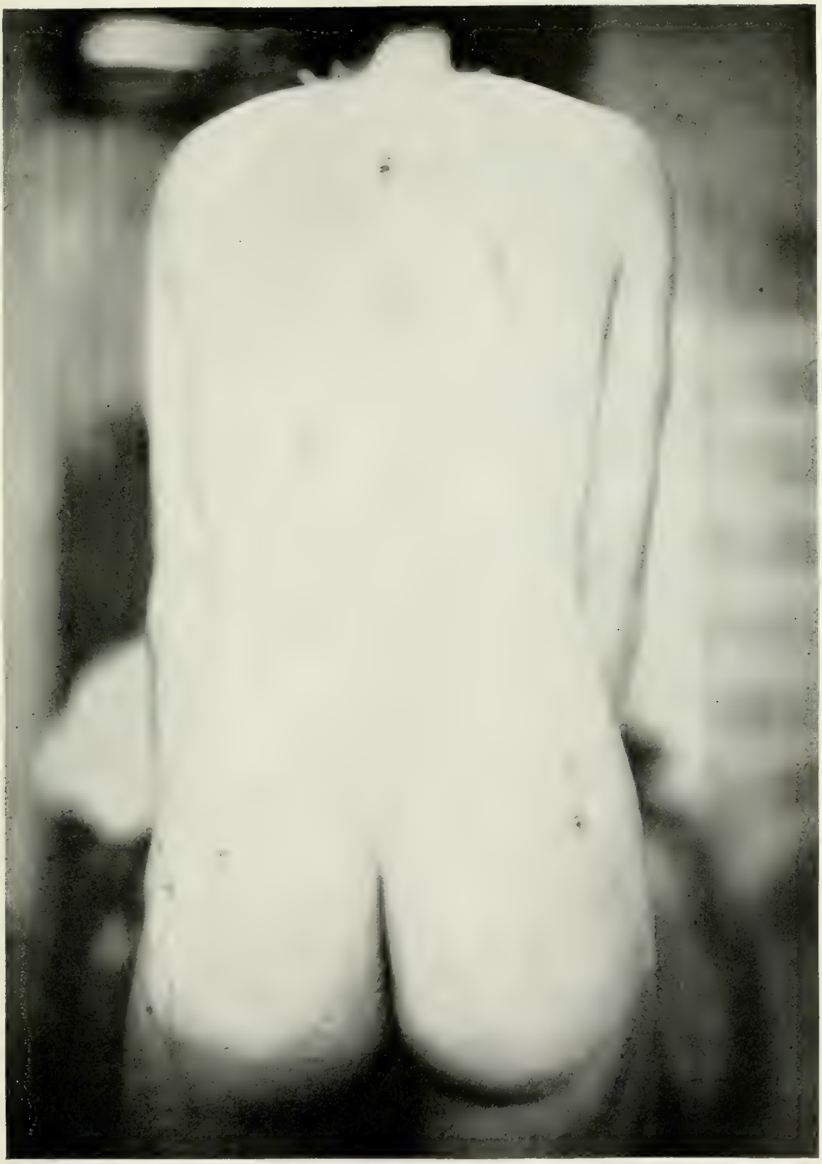


Fig. 4.—Case V. Tertiary syphilis resistant to mercuric treatment.

month by a sore throat and a roseolar eruption on the chest, abdomen, face, back and extremities. He had headaches and a fever and felt weak and tired. He has lost 31 pounds in weight in the past eight months.

The treatment was begun energetically with mercurial inunctions. His specific symptoms did not yield and he developed a dermatitis and a stomatitis. He was then put on subcutaenous injections of mercury on which he improved. Three months after his infection he noticed, while still under hypodermic treatment, four small ulcers on his forehead which healed after a short interval. One month later a bluish red swelling appeared on the forehead which became necrotic in the center and formed a circular ulcer which persists two months later.

During the past two months similar hard lumps and also some serpigenous tertiary syphilides have appeared on the scalp, the cheeks, the back, and the legs, which persist either as indurated places or as ulcers. The progress has been steady from bad to worse in spite of continued treatment with mercury until the present time.

Physical examination showed a general enlargement of the lymph glands and no other lesions except on the skin. The entire body is studded with tertiary syphilides varying from 1 to 5 c. m. in diameter. There are two punched out ulcers on the scalp which are moist and partially encrusted with scabs. The largest ulcer is on the left calf and is secreting considerable pus. Some of the smaller ones are partly healed. The Wassermann was positive but spirochaetes were not demonstrated.

An injection of 0.4 gram of 606 in the form of the sodium salt was followed by a severely indurated and painful local swelling with a general febrile reaction to 103.6° for three days. He required analgesics to secure sleep. The eruptions began to improve at once and at this time, five days after the injection, the patient is sure that his improvement has been more steady than ever before. The ulcers are secreting less and granulating in a more healthy way. The largest one is beginning to cicatrize.

These five cases represent the three principal groups in which Arseno-benzole seems particularly indicated. First, the hereditary cases in which the results are most striking. Second, the primary lesions of which two cases are reported. One (No. 3), a case of slight sclerosis about the chancre, seemed at last interview to be recovering rapidly. The other case is one with a heavy induration at the base of the chancre. The slow improvement even after two injections bears out the conclusions of several observers that chancres in the stage of induration are more difficult to influence with 606 directly in proportion to the size of the sclerosed tissues. The third group of cases is the type which either bear mercury ill or which are not influenced by mercury.

Case IV would under any conditions be difficult of cure and Case V promised to show marked benefit from a single injection after a severe course of mercurial therapy had entirely failed to control the disease.

Case II also would lead one to adopt a larger dosage in large robust men as has been done by Duhot who, as already pointed out, has given as high as 1.1 gram at a single dose and only with benefit. The question of excision of the chancre as an aid to early cure in these cases has also been advised.

These patients were tested further in reference to the excretion of the arsenic by the urine. In four cases tested in this way arsenic was found in the urine within 2 hours of the injection and in two cases which remained in the hospital long enough for observation, the arsenic had disappeared from the urine after 14 and 20 days respectively. Whether these facts have any bearing on the duration of the influence of this method of a single dosage or on the period as to when a second dose should be given if at all, must be decided by a larger clinical experience than is afforded by five cases.

It is possible to report, four weeks after reading this paper, that Case I of hereditary syphilis and Case V of the very resistant tertiary type are free from visible manifestations of the disease. The skin lesions in both cases are healed. Case IV is not improved, and Cases II and III are free from chancre induration, but some glandular swelling and some cutaneous macular eruption still persist in Case II, the one which received the two doses of the drug.

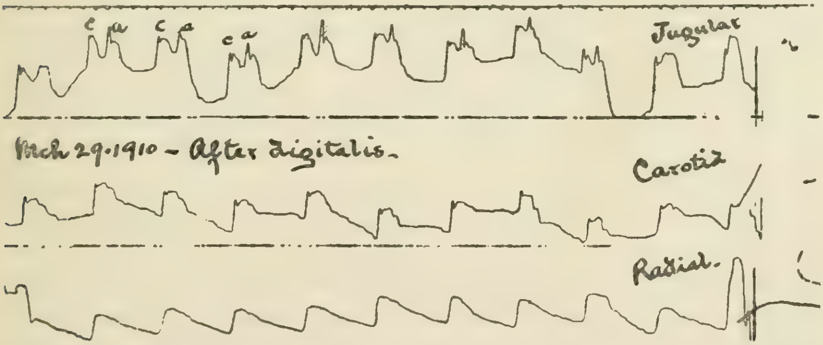
The general conclusion to be drawn from these cases is an endorsement of the universal verdict of medical observers, that Ehrlich has added a remarkable remedy to the treatment of syphilis and has formulated a new idea in parasitic specific therapeutics.

The writer would express his appreciation of the opportunity to study this drug, in advance of its being placed on general sale, to the authorities of the Rockefeller Institute, which has control of its preliminary distribution in America.

REVERSED RHYTHM OF THE HEART, A DIGITALIS MANIFESTATION HITHERTO UNOBSERVED IN MAN.

Van Horne Norrie, M.D. and W. A. Bastedo, M.D.

In his recent Herter lectures on "The Irregularities of the Heart" (January, 1911) Prof. Cushny illustrated reversed rhythm by tracings from dogs, stating that though it was readily produced in these animals by digitalis or aconite, it had not yet been noted in man. The appended tracing therefore becomes of interest, as it was obtained from a patient after a period of digitalis medication. We have gone over the tracing with Prof. Cushny and he agrees that it apparently does indicate a true reversed rhythm.



In the jugular tracing the carotid wave (*c*) precedes the auricular wave (*a*) by a more or less regular interval. As the ventricle is in systole during the beat of the auricle and the auriculo-ventricular valves are closed, the (*a*) wave is abnormally high.

By reversed rhythm is meant a state in which the beat of the auricle follows that of the ventricle instead of, as in the normal condition, preceding it. Cushny was its discoverer in dogs' hearts, and he has advanced the theory that it is dependent upon an excessive irritability of the ventricular muscle with probably some depression of the conductivity in the normal direction along the auriculo-ventricular bundle of His. The result is that the impulse to beat arises in the ventricle instead of at Keith's node, and is conducted along the auriculo-ventricular bundle in a direction the reverse of normal, that is, from the ventricle to the auricle instead of in the opposite direction. In the light of this theory the special interest of this case will be apparent, for it was one of heart block; and if the heart block is due to a destructive lesion of the whole auriculo-ventricular bundle, there could be no conductivity either normal or reverse.

Case notes—Mr. L., aged 68 years, unmarried, born in Germany and of the stolid German type, a veteran of the Civil War and now a laborer by occupation, was admitted to the hospital March 17, 1910. He denied specific disease. During the past five months when walking he had from time to time had sudden attacks of panting and shortness of breath, and a feeling of weakness which required him to catch hold of something for support. At times everything would turn black and he would be oblivious to his surroundings, but he never fell down or had a convulsive seizure. The attacks lasted only a moment and had been increasing in frequency. He thinks that his pulse has been very slow for a long time. His heart was large, the apex not palpable because of emphysema, but percussing five inches from the mid-line in the fifth space, and the right border percussing 2¾ inches from the mid-line in the fourth space. The first sound at the apex was almost replaced by a harsh blowing systolic murmur, which was widely transmitted. The second was barely to be heard, and at a later period was accompanied by a faint diastolic murmur. The pulse was for the most part regular at a rate of 22 to 30, the only exceptions being that on the day of admission one of the house-staff observed an occasional interpolated beat, and on one occasion a nurse recorded a pulse of 48. The brachial, radial and temporal arteries were thickened. The auricular rate, as figured from tracings, was usually in the neighborhood of 64. The breathing on admission was asthmatic, the liver edge just palpable, the spleen not enlarged. The urine was normal. The leucocytes numbered 8,800, with a differential count of polynuclears 73, lymphocytes 25, and eosinophiles 2. The temperature range during his stay in the hospital was 96.4° to 98.4° F.

For five days he was kept in bed without medication, and after the first two days (as recalled later by the patient) had no faint spells. On March 22nd he was ordered powdered digitalis, two grains four times a day. Two or three days later, while still kept in bed, he began again to have spells, though did not report them at first as they lasted such a short time and caused no great inconvenience. But as they increased daily in frequency he complained about them, and on the eighth day of its administration the digitalis was discontinued. The next day he had only two spells, and on the following day, the second after the digitalis administration ceased, had no spells at all.

On the eighth day, while we were feeling the pulse, its rate being 28 and regular, it suddenly became more rapid and was found to be beating at the rate of 56. After three or four minutes there was a long pause, there being no ventricular pulse for nearly a minute; then it resumed its previous rate of 28. During the pause the patient felt faint and dizzy, but recovered promptly on the re-establishment of the pulse. He then remarked that this was one of his usual spells. Shortly afterwards, without warning, the same succession of events occurred with another spell. Then for a long period there was no spell, so, fearing that we might fail to secure a record of the phenomenon, we induced him to wave his arms and rise to his feet, and, luckily, a few moments later, obtained the accompanying tracing. Unfortunately the paper ran out just before the long pause, so of this we have no graphic record. Tracings taken within a few minutes of the published one, while the ventricle was beating at its slow rate, showed varying relations between the auricular and ventricular rhythms, and these, with tracings taken at other times, indicated a condition of complete heart-block.

The items of interest are that with the digitalis medication the faint and dizzy spells reappeared and increased in frequency, but disappeared with the stoppage of the digitalis; that these spells were found to accompany a long cessation of the ventricular beat; that, for a few minutes preceding the cessation of the beat, the rate of the ventricle was double its usual rate and the heart showed "reversed rhythm."

ARTIFICIAL DILATATION OF STOMACH AND COLON AS AIDS IN ABDOMINAL DIAGNOSIS; WITH A HELPFUL SIGN IN CHRONIC APPENDICITIS.*

W. A. Bastedo, M.D.

While we owe much to the laboratory for assistance in abdominal diagnosis, we have still certain measures, not yet in common use, by which we may increase the value of our own bedside observations. Among such are dilatation of the stomach, or of the colon, or both.

When both of these viscera are dilated with air or gas, the abdomen is fairly well split into two regions, an anterior and a posterior, separated by a layer of air or gas (see illustration). We can, therefore, by the dilatation, not only determine accurately the position of stomach and colon, but also locate and determine the relations of an abdominal mass, through its relation to the dividing layer of air or gas.

It goes without saying that the dilatation should always be preceded by the examination of the undilated abdomen.

Dilatation of the Stomach.—To dilate the stomach, about half a level teaspoonful of tartaric acid dissolved in two or three ounces of water is administered, followed in a moment by a solution of similar strength of sodium bicarbonate. The carbon dioxide liberated distends the stomach. If the stomach appears to be one of small capacity, or if there is suspicion of pyloric obstruction, only half the bicarbonate solution should be given at first for fear of overdistension, the rest being given a moment later if the first distension is insufficient. In routine dilatation of a very large number of stomachs by this method, we have noted over-distention in only four, one of them a case with small stomach, the others with obstruction, and in all these too large amounts of sodium bicarbonate were used. Because of the abdominal resistance of the patient, and the comparative awkwardness of the method, we have abandoned, as routine practice, the dilatation of the stomach by injection of air through a stomach tube.

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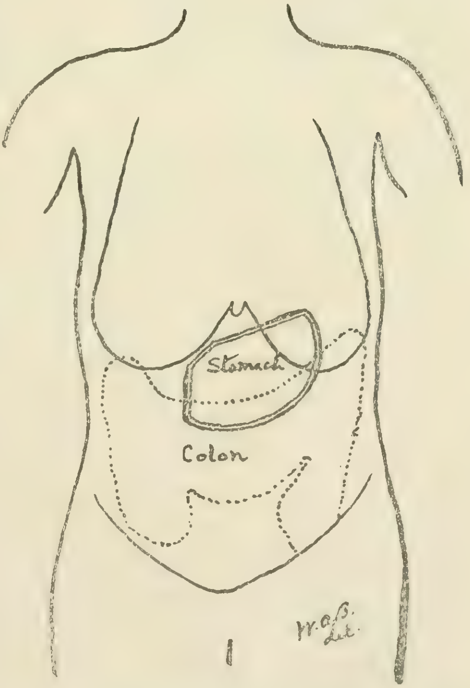
*Read before the Medical Society of the County of Richmond, March 10, 1909.

Dilatation of the stomach is useful in determining the following:

The Size and Position of the Stomach.—With a lax, thin-walled abdomen the dilated stomach appears clearly outlined; with a flat or tense or capacious abdomen, inspection fails, and determination of the stomach limits requires simple percussion, auscultatory percussion in any of its several varieties, succussion, simple palpation if the stomach is thick and hypertrophied, or Glénard's method of palpation when the abdomen is lax. In Glénard's method, when the patient takes a deep breath the ends of the fingers are dipped suddenly to catch the stomach against the vertebral column and are drawn downwards; the gurgle under the fingers and the stomach's lower border are often distinctly felt.



Area of tympany when the stomach (double line) or the colon (dotted line) is dilated.

We do not attempt accurate location of a stomach without a preliminary dilatation, for if the stomach is empty it may give a small area of tympany, even limited to Traube's space, while if it contains food it may percuss dull. Many of the X-ray pictures show contracted stomachs and thus deceive.

In cases where the stomach and intestinal tympany cannot be satisfactorily distinguished, the patient may be stood up and the dull area (seldom flat) of the liquid in the stomach located by percussion. Especially is this necessary, when, owing to the intestines being full of gas, the dilatation of the stomach pushes them out, and results in a bulging of the abdominal wall not only over the stomach but over the intestines as well, and gives one the impression that the whole prominence is stomach.

The Muscular Condition of the Stomach.—Good left to right peristalsis is frequently set up by the dilatation, or by the subsequent examination; or it may be elicited by vigorous tapping over the stomach. Its presence practically rules out atony or myasthenia; its absence as indicating atony is of value only if it persists at repeated examinations. Marked peristalsis always suggests the possibility of pyloric or duodenal obstruction with hypertrophy of the muscular coat of the stomach.

The Location and Permeability of the Pylorus.—We have seen a number of pyloric tumors to the left of the mid line, and at least two at some distance below the umbilical level. These have usually been taken to be in the anterior wall of the stomach or in the colon. But in each of these cases dilatation of the stomach has determined the real site of the tumor by throwing the wandering pylorus into the normal pyloric region. The position of the pylorus, especially if there is stenosis, may also be indicated by palpable vibration as the gas or fluid passes through it, or by the loud, close-to-the-ear gurgle on auscultation. Peristalsis with absence of pyloric gurgle indicates complete closure of the pylorus; and this is usually due to pyloric spasm whether associated with an organic lesion or not. One gastro-enterostomy case sent to us for observation showed this marked peristalsis towards the pyloric region without pyloric gurgle; and we were puzzled till we learned that the pylorus had been completely removed, and a gastro-enterostomy had been done too far to the left. It is to be remembered that where pyloric obstruction is suspected, the sodium bicarbonate solution should be used cautiously.

The Location of a Tumor of the Upper Abdomen.—Of a wandering pylorus we have just spoken. Dilatation of the stomach throws forward tumors of the anterior abdominal wall, of the anterior wall of the stomach, of the greater curvature, of the gastro-colic omentum and of the left lobe of the liver, and in consequence these become more distinct, or at least not less distinct; while the dilatation throws backward and causes to disappear tumors of the posterior stomach wall, of the lesser curvature, and of the pylorus, as well as tumors posterior to the stomach, as of the pancreas, left kidney, or transverse mesocolon. We have had two cancers of the gastro-colic omentum which were well pushed forward by the dilatation; two tumors which were diagnosed as tuberculous omental tumors, but which disappeared with the dilatation and were both found to be cancers of the posterior gastric wall; three diagnosed to be in the enlarged left lobe of the liver but found to disappear with the dilatation, and to be, one a cancer of the pancreas, and the other two cancers of the lesser curvature; and other tumors of similar import.

The Location of the Site of Tenderness.—The same rules apply as in the location of a tumor. A patient complains of soreness in the stomach region. On palpation no tumor is felt, but there is a tender area; after dilatation the tenderness cannot be elicited except on very deep palpation, and after the gas has disappeared from the stomach the tenderness returns. In such a case the tenderness must either be in the posterior gastric wall, or behind the stomach. A few cases in which pain at the same spot resulted each time the stomach was dilated, at intervals of several days, were thought to have chronic

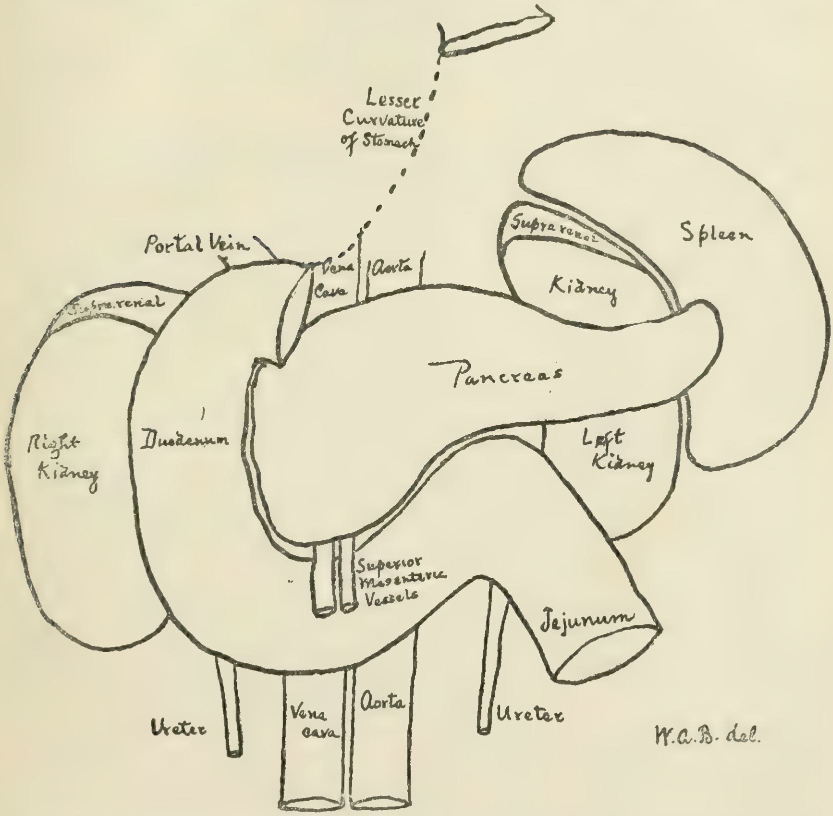


Diagram after Gray and Quain illustrating the important organs immediately posterior to the stomach and colon.

ulcer with adhesions, and in some there was a good ulcer history; but none of these cases have come to operation, to confirm or upset the diagnosis.

Dilatation of the Colon.—To dilate the colon a soft rubber rectal tube is inserted about twelve inches and air forced through it by means of an atomizer bulb. The introduction of the tube is facilitated if air is forced through it during its passage up the rectum.

Dilatation of the colon, or the attempt to dilate it, is useful in determining the following:

Rectal Obstruction.—If the rectal tube cannot be passed into the sigmoid flexure, even when the rectum is dilated in advance of the tube by the forcing in of air as the tube is being passed, an obstruction is suggested, and should be looked for with the pneumatic sigmoidoscope. If the tube passes readily we may elicit information as follows:

Colon Obstruction.—An obstruction in the transverse colon is usually easily determined, the failure of the air to pass the obstruction being obvious. But an obstruction at the splenic or hepatic flexure may not be readily located. If the air easily distends the sigmoid without passing into the transverse colon, a splenic flexure obstruction should be suspected. We have had but few cases of splenic flexure obstruction, and in some the test was impaired by marked tympanites; but we have had a number of cases in which splenic flexure obstruction was thought to be present, but was shown not to be so by the easy passage of air into the transverse colon. Hepatic flexure obstruction is still less common, but if the air fills the transverse colon but not the caecum it may be suspected. One case in which failure of the air to pass, and other signs, indicated an obstruction of this kind, proved to have a tuberculous stenosis through which a lead pencil could scarcely be passed.

The Location of the Sigmoid Flexure.—Occasionally the flexure is so long that it becomes pathological. In one of my cases of marked constipation it was found to extend to two inches to the right of the mid line, and from navel almost to symphysis pubis.

The Location of the Transverse Colon and the Condition of Its Muscle.—One may use this dilatation to determine enteroptosis, and especially to distinguish colon from stomach in some doubtful cases. As to the muscular power a flabby non-sensitive atonic colon may become enormously distended without much discomfort; while a colon abnormally sensitive and vigorous will result in either prompt expulsion of the air by rectum, or hard colicky pains.

The Location of the Cecum.—This is frequently bulged so that its outline is clearly visible, or at least is easily determined by percussion.

The Site of a Tumor or Tender Spot.—The dilatation fairly divides the lower abdomen into anterior and posterior regions, pushing forward and making more prominent a tumor or organ of the anterior region, and pushing backward and causing to disappear a tumor or organ of the posterior region. The important anterior structures are the anterior wall of the colon, the adjoining omentum, spleen, gall-bladder, a Riedel's lobe of the liver, and any intra-abdominal mass attached to the anterior abdominal wall. The posterior structures include the posterior wall of the colon, the mesocolon, duodenum, kidney, ureter, aorta and anything else that is retroperitoneal as lymph nodes, psoas abscess, etc.

Therefore, by dilating the colon, supplemented in some cases by dilatation of the stomach, it is possible to differentiate a right kidney from a gall-bladder or a Riedel's lobe, or a left kidney from a spleen, or a tumor of the anterior wall of the colon or a tuberculous omental sausage from a tumor of the posterior colon wall. A few cases will illustrate; (a) A man of 35 years with a slight tuberculous lesion at the apex of the right lung had a slightly movable tender mass two inches to the left and just below navel level. It seemed to several to be tuberculous peritonitis, but on dilatation of the colon it disappeared and as blood was found in the feces, it was diagnosed as carcinoma of the posterior wall of the colon. This diagnosis was confirmed at operation. (b) Patient had been in bed eight weeks with some symptoms of colon obstruction and a palpable tumor in the left flank thought to be either kidney or a carcinoma of the colon. On dilatation the mass moved forward, thus ruling out kidney, and began to present a free edge as of a spleen. Further dilatation resulted in the moving of the mass up beneath the ribs. It was a movable spleen. (c) The next case illustrates what can be done in diagnosis in a busy clinic. A Greek of 50, rather emaciated, with a two-inch incision scar in the gall-bladder region, complained of several weeks' soreness beneath this scar. He spoke no English, and nobody at hand spoke Greek. A rather tender mass, distinctly cystic, had been made out, and as the diagnosis of various observers included cystic kidney, hydronephrosis, pancreatic cyst and gall-bladder he was sent to us for further data. The cyst, felt to be two inches or more in diameter, was centered beneath the lower end of the scar. It could not be moved up and down, and was independent of the respiratory up and down excursions of the liver, therefore was probably not gall-bladder. After dilatation of the stomach the mass was unaffected, but on dilatation of the colon it was pushed forward so that the abdominal wall looked as if it had a small apple beneath it raising it up. It was positively, then, neither kidney nor pancreatic cyst, which would have been pushed backward. But, though immovable up and down, it could be slowly moved from side to side through a distance of two inches, unless the rectus muscle was held tightly, when it could not be moved at all. The case was therefore sent to the hospital as an intra-abdominal cyst attached to the anterior abdominal wall. Repeated attempts by several physicians failed to elicit any hydatid succussion, but the surgeon found an echinococcus cyst attached to the inner sheath of the rectus muscle.

The Presence of Chronic Appendicitis.—In the routine examination of digestive patients by colon dilatation, I noted, some three years ago, that acute pain or tenderness in the region of McBurney's point on dilatation of the colon regularly meant appendicitis. I have now many cases in which the finding has been proved by operation, and in every case operated upon in which the sign was positive, a diseased appendix has been found. I have a few cases thought to be appendicitis in which the sign was negative; in some of these from one to three years have passed without any appendix manifestations; in the others operation was performed and the appendix found normal. In no operation case with the sign negative was appendicitis found. I would ask all surgeons to employ this method so that we may determine its exact value; for it would seem to be especially useful in those cases in which slight tenderness to finger point pressure at McBurney's point, still leaves doubt as to the advisability of operation.

We for a long time believed that the pain and tenderness were due to adhesions, but in one case with much indefinite abdominal distress in which the sign was positive, operation by Dr. Walton Martin showed a four-inch perfectly free appendix that was swollen and inflamed at the end. I surmise that the dilatation of the cecum made pressure on the inflamed area, or on some sensitive nerve-filaments, and so caused the pain and tenderness, but there were no adhesions.

In several cases of right-sided salpingitis and ovarian trouble, the sign has been negative, though a diffuse tenderness low down and extending to the middle line has been present in three or four of them. In one case of salpingitis in which the sign was positive, the appendix was found involved as well as the fallopian tube.

The Presence of Adhesions.—Pain and tenderness are regularly brought out at the site of old abdominal operations, and occasionally in various parts of an abdomen that has not been operated upon. Their occurrence in the same spot at repeated examinations suggests adhesions.

These few examples will suffice to show the usefulness of stomach and colon dilatation, particularly the latter, in abdominal diagnosis.

In closing I might add that I have had a number of obscure epigastric hernias, in which the diagnosis became easy after the dilatation. A recent case will illustrate. A large woman with prominent abdomen and considerable adipose had much pain in the stomach region which became worse as the day progressed and was often relieved only by vomiting. After a day or two in bed she would feel well again, but on getting up was as bad as before. The question of exploratory operation was considered. We saw the case after she had been kept in bed for two days, and after her pain had ceased. On dilating the stomach, and palpating, a vibratory gurgle was felt in the middle line above the navel, and suggested hernia. On cough a little prominence appeared, and finally became as big as one's fist. Operation was performed for epigastric stomach hernia with adhesions, and the patient has had no recurrence of the old trouble.

THE PHYSICIAN'S DEMANDS OF THE PHARMACIST.*

W. A. BASTEDO, M.D.

To an audience of pharmacists and physicians this subject would scarcely seem to need elucidation. Yet in answer to the query, "What do you think of the pharmacist as a professional man?" I have found medical men so ready with adverse criticism of the pharmacist that there must be either on the physician's part a woeful misunderstanding of the pharmacist's work, or on the pharmacist's part a failure to make good. I believe, therefore, that the better pharmacists will appreciate a frank statement of what the physician requires of them, not as analytical chemists, or bacteriologists, or microscopists or dealers in drugs, but as dispensers of prescriptions.

The greatest of these demands is that the pharmacist shall have pharmaceutical ability. The physician must have such confidence in the average pharmacist's ability that he will trust the pharmacist to make up extemporaneously any formula into any kind of preparation desired. Let the physician gain such confidence, and let him understand that the cost of extemporaneous preparations is not prohibitive, and not only will he consider it unnecessary to specify Q. E. D. & Co's pills, or A, B & C's elixir, but he will welcome the facilities at hand to further his therapeutic practice. To establish such confidence the pharmacists as a class must convince the physicians as a class (so that the attitude of confidence shall be the traditional attitude of the physician) that they, the pharmacists, have the ability to make and the facilities for making all kinds of pharmaceutical preparations quickly, and accurately, and according to the methods of up-to-date pharmacy.

The physician of the past seems to have had some hard experience, for he has handed down to his successors a certain traditional lack of confidence in the druggist. The (medical) graduate of a few years ago knew little pharmacy because he was not taught pharmacy, and it was easy for him to drift into the use of ready-mades. But the medical student of today is being taught pharmacy, not to enable him to serve as a pharmacist, but to fit him for proper prescribing; and he will neither on the one hand be a user of the ready-made formula, nor on the other hand will he accept suppositories made by hand, lumpy and large and unsightly, or ointments with a polka-dot appearance from inadequate admixture, or fermenting mistura cretae, or flat, strongly acetic spirit of mindererus. These new medical men will understand and appreciate good pharmacy; and now is the time of all times for the pharmacist to make quite sure that what he practices is good pharmacy.

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*Read at a joint meeting of New York physicians and pharmacists held at the Academy of Medicine, May 19, 1909.

One thing is certain that before he undertakes to win the physician's confidence, the pharmacist must have confidence in himself and in his own pharmaceutical ability. When a pharmacist dispenses a ready-made emulsion or a gelatin-coated pill, such not being specified on the prescription, does he show confidence in his own ability? Does a pharmacist who advises physicians to prescribe capsules rather than cachets because of the difficulty and expense of making the latter, have pharmaceutical ability? And what must we think of an apothecary who is unable to supply a simple tablet triturate at 9 P. M. because he does not have it in stock and the wholesale houses are closed until tomorrow morning? Yet those are instances recorded in my own book of experience.

A second demand of the physician is that the pharmacist shall use due carefulness—carefulness in reading the prescription and carefulness in checking his bottles, his weights, his measures and his label. Mistakes will happen, but, as far as possible, some checking and recording system by which the majority of errors must be detected, should be in common vogue among pharmacists, and understood by the physicians to be the common practice. Such a system should render it well-nigh impossible to have the thick sweet, official rhubarb and soda mixture which contains 35 per cent of glycerin, dispensed on a prescription written "Misture rhei et sodae, U. S. P., sine glycerino"; or the compound tincture of cinchona dispensed for the compound tincture of lavender, these being next door neighbors on the druggist's shelf.

One of the crying evils in this regard, an evil that it seems impossible to overcome in the smaller stores, is the necessity that the dispenser is so frequently under, of leaving a prescription half dispensed while he performs the functions of a sales clerk in some other part of the store.

A third demand of the physician is a proper ethical attitude on the part of the pharmacist. That is, that in regard to the nature of the ingredients or the therapeutic usefulness of a prescription the pharmacist shall refuse absolutely any information to the patient. I might say to the pharmacist that among patients, particularly nervous and spoiled ones, the physician is often handicapped by prejudice against certain drugs, one patient saying, "I can not take cascara;" or another, "All coal-tar products poison me severely;" or another, "Calomel always makes me dreadfully ill for a week." But these same patients do well on rhamnus purshiana, or acetphenetidin, or hydrargyri chloridum mite, and the revealing of the real nature of these substances is a serious matter. A patient recently reported to me that his pharmacist had commended my prescription for rheumatism, but had said that if he had been prescribing such a mixture he "would have added a little fluid extract of ——," something the name of which the patient had forgotten, which was "the best thing yet for rheumatism." Comment on the wisdom of such displays of knowledge (!) is unnecessary.

Obligations of Physicians to Pharmacists.—Still, although without a definite code of ethics. many pharmacists do try to treat ethically both patient and physician, even sometimes at an immediate commercial loss. And we physicians, who make so many errors in our prescription writing, must never cease to be grateful to the pharmacists, who have so often protected us from the consequences of our prescription errors, and have times without number shouldered the unjust blame themselves. Indeed, physicians have many faults that at times are trying to the pharmacist.

The good pharmacists are doing much to improve the conditions of the practice of pharmacy. Through their efforts, and theirs alone, there has been a great advance in the educational requirements of the pharmacist. But the fact remains that the efficient and conscientious pharmacist is suffering for the faults of the inefficient and unscrupulous; for the physician has no satisfactory way of distinguishing them. As to how far we physicians can help ourselves in these matters, our success in improving the milk supply may offer a suggestion. A few years ago there was no satisfactory distinguishing mark between the good and the poor milks. Now we have certified milk, which is produced under conditions laid down by the milk commission of the county medical society—conditions as to care of cows, stabling, milking, bottling, etc. —and which on occasional tests is found to conform with certain high standards. All specimens of milk can not be tested, but it is found that milk coming from dairies so controlled is very uniformly of high quality. For the benefit of pharmacy and of medicine I hope that the day is not far away when we shall have "certified pharmacies," i. e., pharmacies the prescription departments of which are arranged and conducted according to certain practicable specifications laid down by a pharmacy commission of the county medical society, with the advice and counsel of the best pharmacists. The good and conscientious men of the pharmaceutical profession will welcome such distinction; and some of those who are not naturally good will develop a conscience under the new incentive.

To sum up: The things that the physician demands of the pharmacist as a dispenser of prescriptions are: first, pharmaceutical ability; second, responsibility and carefulness; third, a proper ethical attitude towards the prescription and the prescriber. And we might add, a method by which the physician may distinguish the sheep of the pharmaceutical fold from the goats.

CHLORIDE AND WATER TOLERANCE IN NEPHRITIS.

From the Service of Dr. Theodore C. Janeway.

Karl M. Vogel, M.D.

Widal, Achard, and Strauss some years ago called attention to the bearing of sodium chloride retention on the still unsettled question of nephritic edema, and since then regulation of the chloride intake has been found of value, not only in nephritis, but also in the treatment of fluid accumulations of other origins. The measure has been applied with more or less success in cases of cardiac disease, inflammatory exudates, such, for example, as tuberculous peritonitis, in diabetes insipidus, and in conditions in which it is desirable to restrict the amount of fluid ingested. It is chiefly in nephritis, however, that resort to a salt-poor diet has been found useful, and it is becoming evident that while the therapeutic results are not invariably as brilliant as Widal predicted, investigation of the chloride tolerance can furnish valuable diagnostic and prognostic indications. According to Widal, two clinical types of nephritis may be recognized, one in which nitrogen retention predominates, and another in which the permeability of the kidney for sodium chloride has become impaired. Edema, he contends, depends solely on the retention of chlorides, and is largely a physical phenomenon, dominated by the body's intolerance of non-isotonic solutions. By determining, therefore, the fluid intake and output, the sodium chloride intake and output, and the fluctuations of the patient's weight, it should be possible, provided the functional capacity of the kidneys has not been too seriously impaired, to manage the patient's dietary so as to dissipate existing fluid accumulations and guard against their recurrence. In general, these theoretical considerations of Widal's have received abundant clinical confirmation, but unfortunately experience shows that the problem of nephritic edema can not be reduced to quite such simple terms, and instances are not infrequently encountered in which the condition proves refractory even to persistent reduction of the salt intake. Blooker,[1] indeed, who very carefully investigated the question of chloride retention in a series of selected cases, comes to the conclusions that nephritic edema is only rarely the result of primary chloride retention, and that resort to an extremely salt-poor diet is not often necessary.

The elaborate studies of Schlayer and Takayasu,[2] however, on the artificially induced nephritis of rabbits, furnish a very substantial experimental basis for further clinical work in this direction, and leave no doubt as to the interest of the chlorine balance in nephritis from the standpoint of diagnosis and prognosis, as well as of treatment.

These observers found that it was necessary to distinguish clearly between the effects of tubular and vascular lesions. Injury to the tubular epithelium invariably caused great reduction in the capacity for the excretion of sodium chloride, the retention being directly proportional to the intensity of the lesion. Vascular changes, on the other hand, were without influence on the elimination of the salt, and it was found that a low specific gravity urine might be the result of overirritability of the renal vessels, and in this case was not accompanied by any impairment of chloride excretion; or it might be due to tubular lesions and then involved chloride retention which might reach the highest grades.

Clinically, there are, of course, many transitions between these two types, accordingly as the tubular or the vascular lesion predominates, but some insight into the underlying conditions may certainly be gained by watching the sodium chloride excretion, and particularly the manner in which known amounts of salt added to the diet are disposed of. In other words, just as it is essential to determine the carbohydrate tolerance of a diabetic, or the purin tolerance of a gouty patient as a preliminary to proper treatment, the ability of the nephritic to eliminate sodium chloride and water must be investigated in order to secure a basis for the intelligent management of his disease. According to the information so elicited a diet with no restriction as to salt content, or one that is salt-poor, or, in extreme cases, one that is as nearly as possible salt free, is employed, and the patient is subjected to no more hardship in the matter of salt abstention than the necessities of his case demand. At the same time, the manner in which the kidney deals with known amounts of sodium chloride and water furnishes, at least in some measure, an index of its functional capacity in general, and aids in establishing the anatomical diagnosis. It is, therefore, apparent that whatever may be thought of the salt-poor diet as a routine mode of treatment, the indications to be gained from a study of the chloride and water intake and output are sufficiently valuable to render it advisable to make such determinations in beginning the treatment of every case of nephritis that is at all severe. The following three cases are cited briefly as an example of the behavior of the chloride balance in different types of the disease.

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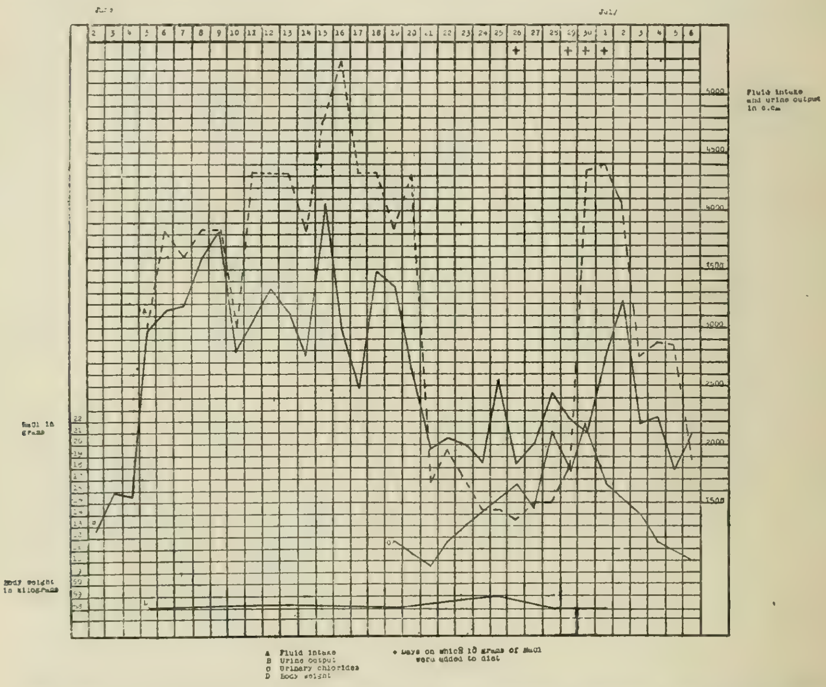
[1] Blooker, J. W., Deutsch. Arch. f. klin. Med., 1909, xcvi, 80.

[2] Schlayer u. Takayasu. Deutsch. Arch. f. klin. Med., 1910, xcviii, 17.



Case I*.—Patient T. L., No. 81341. An American carpenter, aged 27 years, admitted to the service of Dr. Norrie in St. Luke's Hospital, on June 2, 1910. The patient's family and previous history are negative. He takes two cups of tea and one of coffee a day. He uses no alcohol and is a moderate smoker. Present illness: One month before admission he caught cold, had a sore throat, a chill, and some fever. His doctor said he had grippe. Two weeks before admission his legs became swollen, remained so for a week, and then the swelling disappeared. His face was puffed at the same time. He urinates very frequently, especially during the night. The urine is very dark colored, rather reddish sometimes. No visual disturbances. He has slight headache. Chief complaint: Swelling of legs and frequent micturition.

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*Chloride determinations by Mr. G. M. Goodwin, Columbia University, 1911.

Physical examination: the patient is a well nourished young man without dyspnea, cyanosis, or edema. He is moderately prostrated, his skin is pale and the tongue coated. The heart is not enlarged, there are no murmurs, and its action is regular and forcible. The second aortic sound is slightly accentuated. Pulse: slight increase in tension; the vessel walls are palpable but soft. No edema. On admission, the blood pressure was 160; it fell in four days to 130; and after that varied between 110 and 130. On admission, the patient's urine contained 10 per cent of albumin by volume. A few hyaline and granular casts, a few leucocytes, and also some epithelial casts were found. The specific gravity varied between 1.008 and 1.020. After May 26, no more than a very faint trace of albumin was found.

The patient received no drugs and was on regular diet. During the period from June 5 to June 20 he was given very large amounts of water which as the chart shows, were well excreted. From June 21 to June 29 he was told to follow his inclinations in regard to the amount of water taken, and from June 30 to July 2 his fluids were again increased. On June 26, 29, and 30, and July 1 he was given an additional amount of 10 g. of sodium chloride daily. This was followed by no increase in weight, and during the days following the administration of the extra salt the excess was completely excreted. During four days on regular diet the average excretion of sodium chloride was 11.4 grams. During eleven days in the course of which 40 extra grams of salt were given the total excretion of sodium chloride was 149 grams. Deducting the 40 extra grams, the average excretion for this period would be 13.4 grams, showing that the kidneys were able to excrete quantitatively all the excess. though its elimination was somewhat delayed.

Case II*. Patient C. H., No. 82055. A German butcher, aged 44 years, who was admitted to the service of Dr. Janeway on July 9, 1910. The patient's family history was irrelevant. He had had measles in childhood, Neisser infection twice in early youth, and lues 10 years ago. He takes six glasses of beer a day and some whiskey, and smokes six cigars. His general health has always been good. Present illness: Six or seven weeks ago he noticed that his feet and legs were getting swollen, and later on his thighs, abdomen, and scrotum developed the same condition. At times there was puffiness about the eyes. He continued to work until three weeks ago, when he went to bed and has remained there most of the time ever since. His physician examined the urine frequently and told him he had kidney disease. Chief complaint: Swelling of abdomen and legs.

Physical examination: The patient is a large, well nourished man. He does not appear prostrated, shows very slight dyspnea and no cyanosis. The pupils are equal and react to light and accommodation. The tongue is coated; the teeth are in good condition. The apex impulse is not seen, but is faintly felt in the fifth space, five inches from the midline. The right border percusses one inch to right, the left border five inches to left. The sounds are of good quality; no murmurs nor accentuations are made out. Heart action is regular. The pulse is of fair force, the tension 125 mm. Vessel wall not felt. Lungs: at the right base posteriorly there is dulness from the angle of the scapula down, with diminished breath sounds and voice. Fremitus comes through. The abdomen is soft, the liver and spleen are not felt. There is marked edema of both legs, extending slightly up as far as the thighs.

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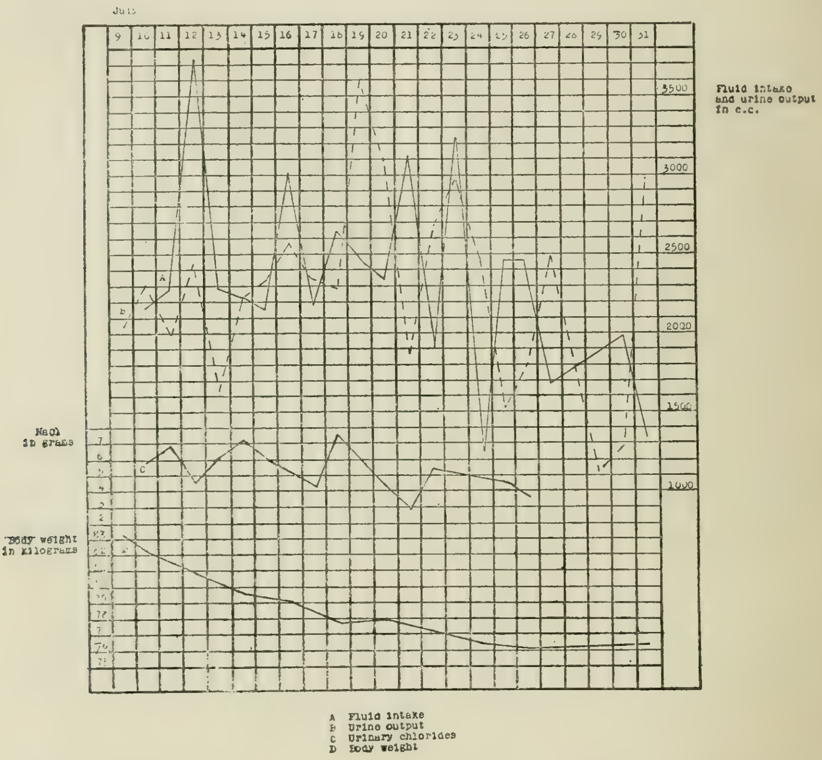
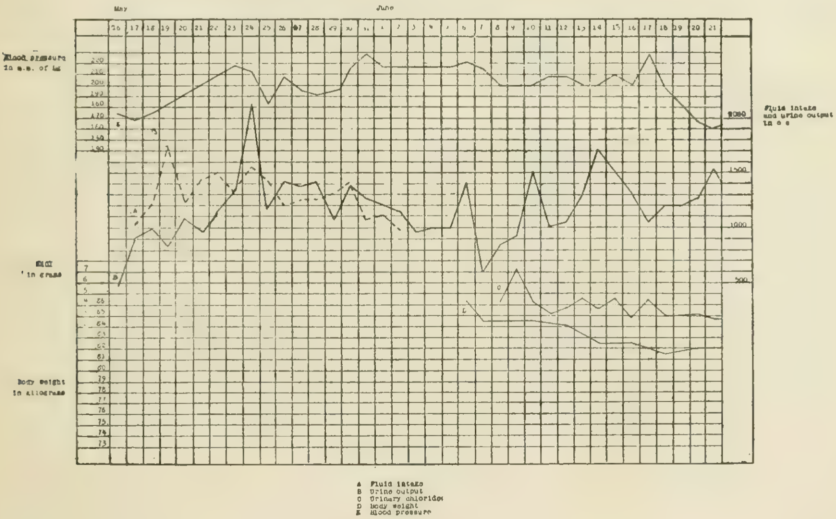
*Chloride determinations by Mr. Harbeck Halsted, Columbia University, 1911.



On admission, the patient's urine contained 30 per cent of albumin by volume, with a few hyaline casts. On July 12 it contained 60 per cent of albumin, and after that date the amount steadily decreased until at the time of discharge there was only a trace. The specific gravity was 1.020 to 1.024. There was no sugar. Hyaline casts were always present. The diet was the hospital salt-poor diet (containing 3.25 g. of sodium chloride per day) with no restrictions as to the amount of water taken. No drugs were given, except an occasional cathartic. The edema steadily decreased, at first rapidly, later more slowly, but on August 1, when the patient was allowed to leave his bed, there was very slight edema left over both tibiae.

In this instance, in which no treatment was resorted to except the diet and the rest in bed, in a little over three weeks the patient's weight fell from 83 to 76 kilograms; his edema disappeared; and the chloride output was greater than the intake. The kidneys are apparently still fairly permeable to sodium chloride, and there is a strong likelihood that careful management of the patient's chloride metabolism would do much to prevent further exacerbations.



Case III.*—Patient A. J., No. 81447. An Irish laborer, aged 50 years, sent into St. Luke's Hospital by Dr. A. W. Hollis, and admitted on May 16, 1910, to the service of Dr. Norrie.

History: The family history is irrelevant to the present condition. Past history: For the past fifteen years he has had "rheumatism" every winter. Formerly the attacks were accompanied by pain, but of late there has been only swelling and impairment of function. Luetic infection about thirty years ago, treated and said to have been cured. Neisser infection denied. Infection of a finger 12 years ago. Personal history: General hygiene good. Takes tea twice a day, no coffee, and until the beginning of present illness eight to nine glasses of beer daily. For the last two years his appetite has been poor, but otherwise the patient has felt well. Present illness: About one month before admission, he began to suffer from anorexia, nausea, and morning vomiting, which he relieved by taking a glass or two of whiskey. Three weeks before admission his legs began to swell, there was puffiness of the left cheek and eyelids, and edema of the scrotum. No pain or urinary symptoms. Ten days before admission a severe headache lasting three days compelled him to stop work for two days. Some days before admission he "caught cold," had two chills, coughed with slight expectoration, and suffered from shortness of breath. During the past month he has been able to work only at intervals. Chief complaint: Swelling of legs, shortness of breath, and morning vomiting.

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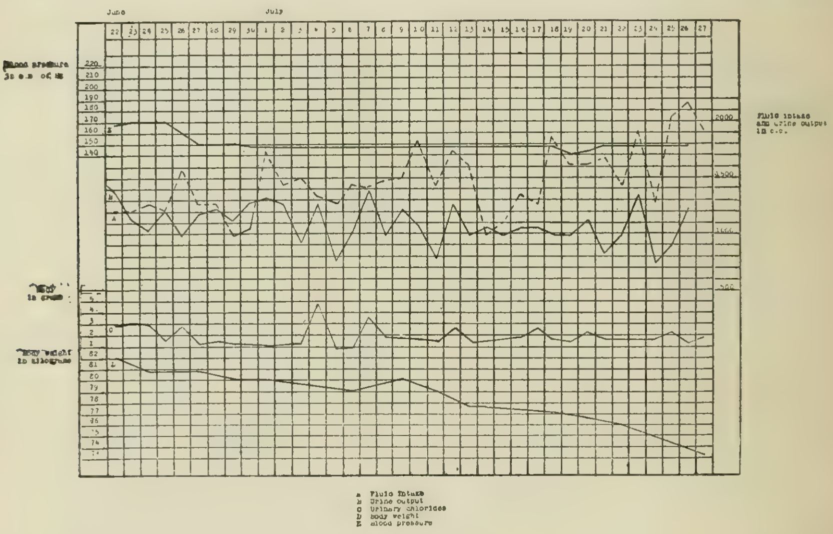
*Chloride determinations by Mr. John A. Vietor, Columbia University, 1911.

Physical examination: The patient presents moderate edema of the lower extremities and about the trunk. There is slight cyanosis of the finger tips but no dyspnea. The apex beat is in the fifth interspace, six inches from the midsternal line. No superficial impulse except in the epigastrium. The sounds at the apex are distant, but of good quality; second pulmonic sound faint; second aortic sound accentuated. The action is regular and forcible; pulse regular, of good force; the vessel wall is markedly thickened; blood pressure, 176. At the apex of the left lung there is dulness, and many subcrepitant rales are heard immediately below the clavicle. Over both sides in front, sonorous and wheezing expiration; sibilant and sonorous breathing over the posterior chest. At both bases marked dulness, on the left side extending almost to the angle of the scapula. Fremitus, voice, and breathing are diminished. Coin sound is heard on both sides. The liver percusses slightly large, but its edge can not be felt. Spleen is not felt.



During the patient's stay in the hospital his urine usually showed a specific gravity of 1.020 and an albumin content of from 10 to 80 per cent by volume. There was no sugar. Many hyaline and granular and a few waxy casts were found. On June 24, the left chest was tapped, and 1,560 c. c. of fluid removed. On July 11, the aspiration was repeated, and 1,740 c. c. of fluid, containing 19.1 grams of NaCl were removed. At a third aspiration on July 26, 2,040 c. c. of fluid with 14.3 grams of NaCl were removed.

The treatment comprised the administration of digitalis, short periods on glonoin and diuretin, and hot packs, given from June 21 to July 11. On June 6 the hospital salt-poor diet was begun. On August 1, the patient came under the charge of Dr. Hollis, and a week of regular diet begun on August 31 was accompanied by a gain in weight of 4 kilograms with the appearance of edema in the legs. A return to the salt-poor diet caused a prompt fall in weight, and on September 29, the patient was discharged weighing 73 kilograms, with slight edema of the legs and signs of fluid in the left chest.

The changes in the patient's condition observed while under the salt-poor regime may be summarized as follows, without of course attempting to ascribe them to this particular factor of the treatment: The blood pressure dropped from the neighborhood of 200 mm. to the neighborhood of 140 mm., and remained there. The weight fell from 86 kilograms to 73 kilograms, the weight of the chest fluid removed at three tappings amounting to about 5 kilograms. The urine quantity remained fairly constant between 1,200 and 1,500 c. c. The urinary chlorides fell from about 5 grams to about 2 grams, showing a retention of something over 1 gram daily. This amount was probably accounted for by the amount stored up in the pleural effusion, as the fluid removed in the two instances in which the chlorine content was determined represented about 33 grams of sodium chloride. In the case of this patient, therefore, while a considerable reduction in edema was effected, still it was slow and not sufficient, as shown by the recurrence of the pleural effusion; and there was evidently pronounced impairment of the excretory capacity of the tubular epithelium.

In the following table the NaCl content of some of the commoner foodstuffs is given. The figures are from Strauss,* and apply to the substance in its raw, unprepared state.

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*Strauss, H. Praktische Winke für die chlorarme Ernährung. Berlin, 1910, S. Karger. This contains very complete tables of analyses as well as many useful directions for the preparation of salt-poor diets. The most recent exposition of Widal's views, as well as a good bibliography is to be found in Widal and Lemierre: Die diätetische Behandlung der Nierenentzündungen. Ergebn. d. inn. Med. u. Kinderh., vol. iv, p. 523, Berlin, 1909, Julius Springer.

	Per cent.		Per cent.
Mutton,	0.17	Gelatine,	0.75
Veal,	0.13	Eggs, whole without shell,	0.21
Veal kidney,	0.32	Milk,	0.16
Calves' liver,	0.14	Cream,	0.13
Beef, lean,	0.11	Butter, salted,	1.0 to 3.0
Pork, lean,	0.10	Butter, unsalted,	0.02 to 0.21
Trout,	0.12	Swiss cheese,	2.0
Codfish,	0.16	Edam cheese,	3.30
Salmon,	0.061	Bread, wheat,	0.18
Sole,	0.41	Bread, rye,	0.18 to 0.59
Halibut,	0.30	Oatmeal,	0.29
Mackerel,	0.28	Celery,	0.25 to 0.49
Duck,	0.14	Cauliflower,	0.05 to 0.15
Goose,	0.20	Tomatoes,	0.11
Chicken,	0.14	Cabbage,	0.11 to 0.44
Turkey,	0.17	Lettuce,	0.12
Oysters washed in fresh water	0.52		

Most other cereals, vegetables, fruits, and nuts, less than 0.1.

REPORTS OF WARD CASES.

From the Service of Dr. Austin W. Hollis.

Norman E. Ditman, M.D.

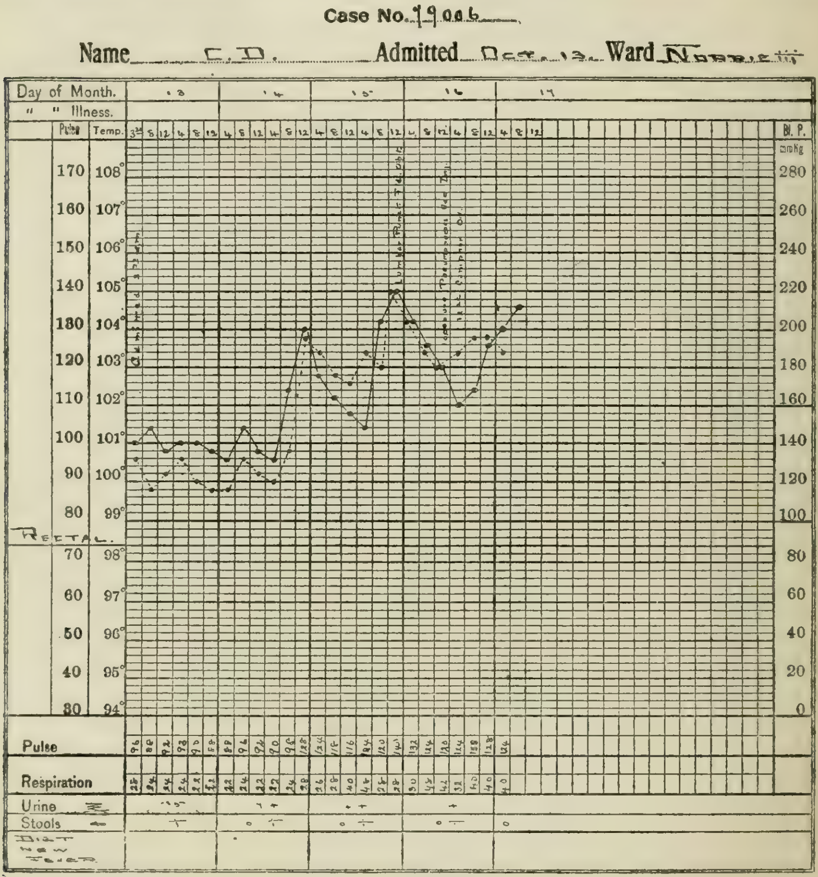
Edema of Brain (Wet Brain) Secondary Pneumonia.—C. D., Retired railroad man, age 63. Family history—Negative. Personal History—Drinks tea and coffee moderately. Has always been a heavy alcoholic drinker. Never drank to excess until 10 years ago. No venereal history obtainable.

Past Illness—Operation for fistula in ano one year ago. Periodical intoxication for past ten years. Gradual failure of health with loss of appetite, indigestion and weakness. He has been very nervous.

Present Illness—For about six weeks the patient has suffered from headache. This has been constant and of a severe type. Ten days ago he fell to the floor while walking. He was picked up in a dazed condition and put to bed. Next day he was well enough to be up. He said he had had an attack of vertigo. He was very nervous and weak and his hand shook so that he could not raise a teacup. He had no appetite. Six days before admission to the hospital he was compelled to go to bed. At that time he complained of headache, chilly feelings and weakness. He became slightly irrational and would get out of bed when not watched and lie down on the floor. He was often confused but recognized members of his family. The night before admission he became entirely irrational, and would not converse with his family. At times consciousness is regained and he becomes restless.

Chief Complaint—Headache and loss of appetite.

Physical Examination—A man of slender frame, poorly nourished. He was irrational on admission to the hospital and when not disturbed lay quietly mumbling. He would not answer questions or follow directions and resisted all passive motions. Skin of good color, numerous small naevi, mucous membranes of fair color. Pupils of the eye are equal, small and react slightly to light. Tongue is slightly coated, tremulous and moist. Superficial lymph nodes not enlarged. Chest—Percussion note is boardy. He cannot be made to breathe deeply and breath sounds are scarcely audible. No altered breathing or râles are heard. Heart—Right border not made out. Left, inside nipple line. Sounds of fair quality without murmurs. There is slight accentuation of the second sound. Pulse regular, small, fair force, some increase of tension. Vessel walls are thickened. Abdomen—Liver percusses to free border. Attempts to palpate are resisted.



Spleen not felt and does not percuss large. No masses or tenderness made out. Extremities—Reflexes normal. No edema. Subsultus marked.

On admission the blood examination showed—leucocytes 4,600. Polynuclear leucocytes 73%; lymphocytes 27%. The Widal test was negative. The urine contained faint traces of albumen and sugar with some hyaline casts and epithelium.

Subsultus, and an active delirium continued, and on the third day after admission there was moderate rigidity of the neck, which increased steadily. By lumbar puncture 8 c.c. of cloudy, pale pink fluid was obtained, which was negative on examination. The fourth day after admission signs of consolidation were detected in the left lower lobe. Ten million pneumococci in a vaccine were injected. Colon irrigations were given daily, sedatives for the delirium, and stimulants in the form of strychnine sulphate, ergotol mxv. every four hours by hypodermic injection, whiskey and camphor. The restlessness increased, the pulse became weaker and more rapid, and on the fifth day after admission the patient died.

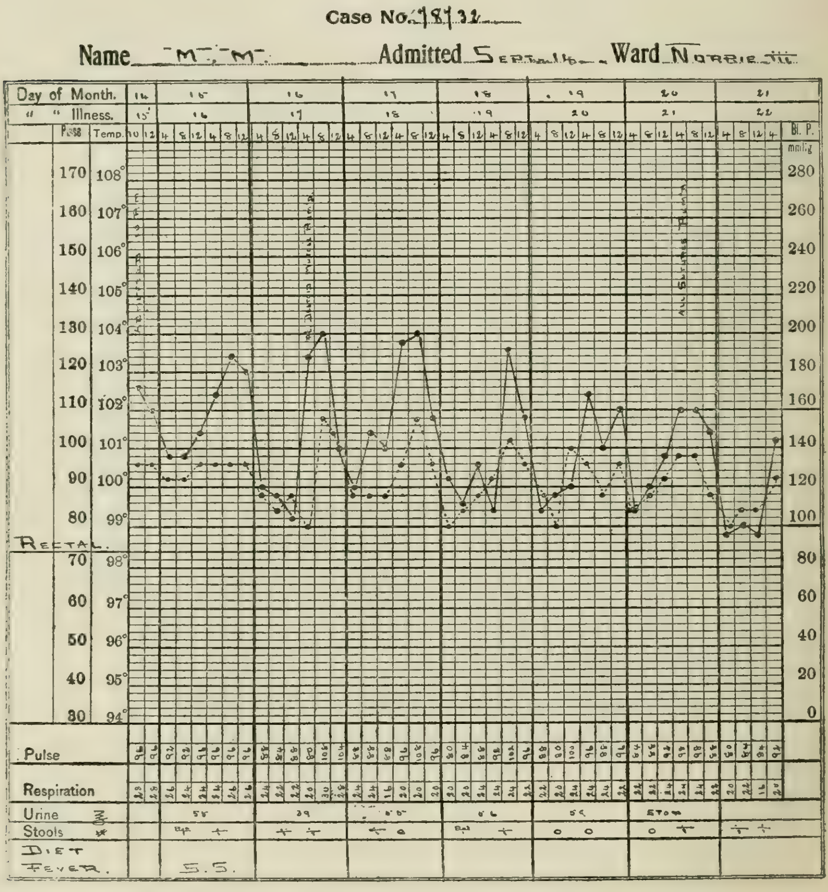
Autopsy Record—No. 794. Heart—Slight atheroma of chordae tendinae, aortic cusps, in sinus of Valsalva and at entrance of the coronary arteries. Lungs—Slight fibrinous deposits on left pleura. Right pleural cavity partially obliterated by adhesions. Consolidation of right lower and middle, and left lower lobes—gray hepatization on the right, red hepatization on the left. Stained smears show pneumococci. Bronchial lymph nodes not enlarged. Liver—Hard and nodular. Normal size. Evidences of connective tissue formation. Kidneys—Normal size. Reddish in color, slightly congested. Capsule slightly adherent. Cortex not narrowed. Markings well preserved. Pancreas—Weight 85 gms. Firm in consistency and apparently fibrous. Spleen—Small (3 oz.) and soft. Some perisplenitis. Suprarenals, prostate, stomach and intestines normal. Brain—Pia is extremely edematous, most marked and chiefly limited to upper surface of hemispheres. No excess of fluid in the ventricles. Substance of cerebrum normal.

Anatomical Diagnosis—Double lobar pneumonia, old pleurisy on right side. Alcoholic wet brain. (Edema of pia.) Cirrhosis of liver. Fibrous pancreatitis. Moderate chronic nephritis. Atheroma of aorta.

Trichinosis—M. M., a man 27 years of age, a German by birth and an electrician. Family history—negative. Personal history—He drinks coffee and beer moderately, smokes tobacco, works hard and keeps good hours.

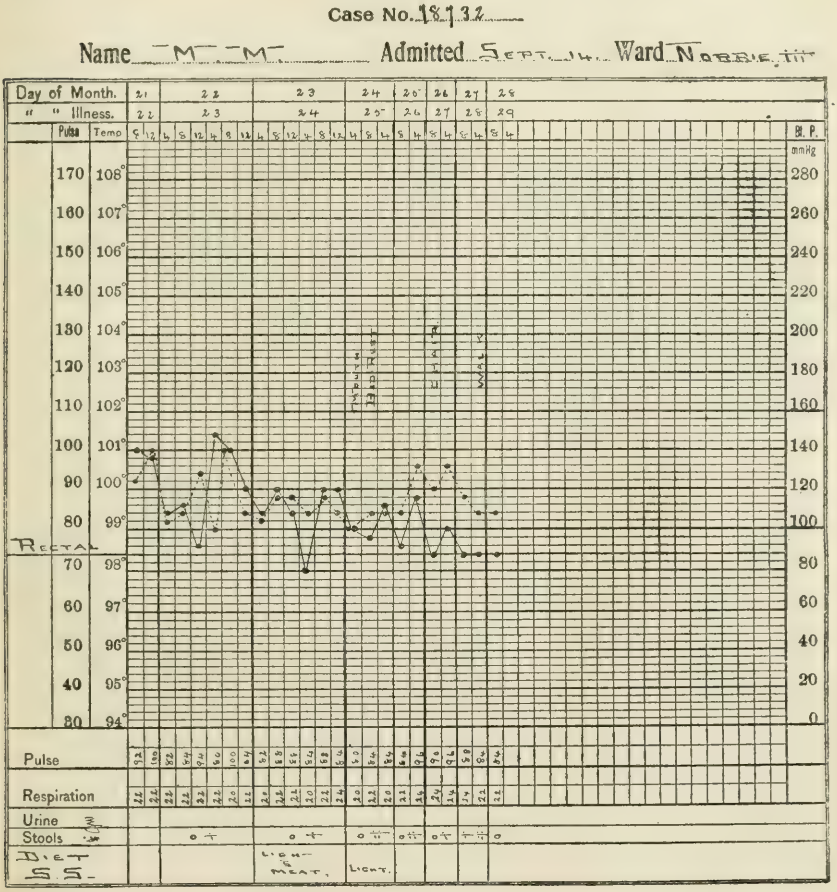
Past Illness—No venereal history. He had a slight attack of rheumatism four years ago.

Present Illness—On the 4th or 5th of September, about nine days before his admission to the hospital, the patient ate some pork in an eastside restaurant. On September 6th he had indigestion and took a laxative. Two days later he had another attack of indigestion. He had severe headache and felt weak and dizzy. He had some fever. These symptoms continued until on September 12 he felt pains and tight feelings in the muscles of his legs. These became worse so that he had great difficulty in walking. Subsequently he had the same sensations in the muscles of his face. His jaws opened with difficulty. His face became swollen so that his eyes protruded. This swelling soon diminished. The muscles in his arms also feel "tight." His appetite and digestion are good and his bowels regular.



Chief Complaint—Weakness, and tightness of muscles.

Physical Examination—The patient is a large and well nourished man, moderately dull and apathetic without dyspnoea. Skin and mucous membranes are a good color. Over the chest and back are several large red patches, slightly raised, and showing some scaling. There are also a few brown spots. None are typically roseolar.



Eyes—Right pupil larger than the left;—both react normally. Tongue is slightly coated. Chest—Respiratory excursion good, percussion note resonant. Breathing of vesicular type with a few subcrepitant râles at the bases. Heart—Normal in outline, sounds of good quality, no murmurs. Pulse regular, full, force moderate, not dicrotic, low tension. Abdomen—Liver descends just below the edge of the ribs where the edge can be felt. The spleen is moderately firm and descends ½ inch below the costal margin. There is no distention, tenderness or rigidity and no masses are made out. Extremities—Reflexes normal. No edema.

At the time of his admission to the hospital, his temperature was 103.4° in the evening, pulse 96 and respiration 26. There was a leucocytosis of 34,300. The differential count showed polynuclear neutrophiles 41%, lymphocytes 7% and eosinophiles 52%. The urine analysis showed no albumen or sugar, sp-gr. 1016, no casts and a positive diazo reaction. A piece of the deltoid muscle removed for microscopical examination showed the presence of trichina. The fever progressively diminished until it was absent at the end of the second week. Soreness of the muscles of the extremities and itching continued for a week or ten days. The differential leucocyte count changed little during his stay in the hospital, but the leucocytes diminished to 19,000 on the 17th day of his disease.

Treatment consisted of rest in bed on fever and light diet, occasional sponges to reduce fever, cathartics and an anthelminthic (aspidium). At the end of two weeks the patient left the hospital feeling well.

SEPTIC ENDOMETRITIS AND SEPTIC PNEUMONIA FOLLOWING ABORTION. M. W., a woman, age 35, was admitted to the medical service of Dr. Hollis, August 27, 1909 (case No. 78551). Family history—negative. Personal history—Formerly drank a large amount of tea and coffee, but no alcohol. Menstruation always regular. Two children living and well. Four years ago had an induced abortion.

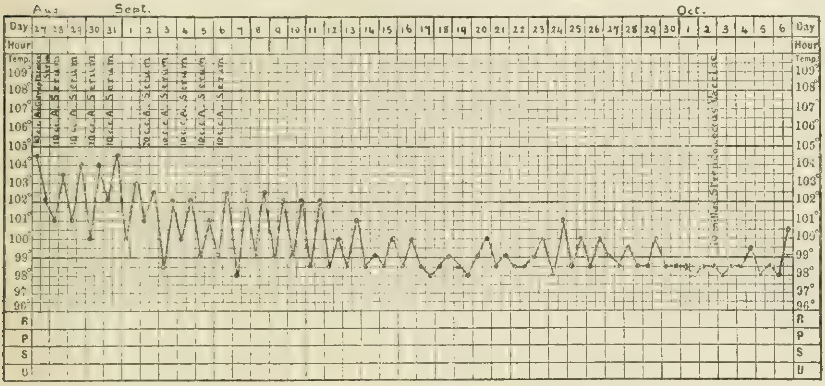
Past Illness—Never sick before.

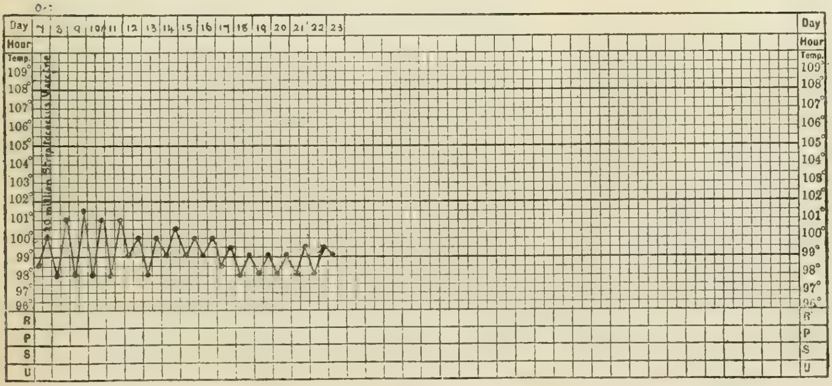
Present Illness—Last menstruation in April. Three days ago an abortion was performed by a midwife by inserting a rubber tube into the uterus. Two previous attempts, which had been made one and two weeks previously, had been unsuccessful. After the last operation she became very ill, becoming so weak that she felt paralyzed. The illness increasing, with vomiting, she was curetted the next day, and the day following she was given a douche. There was no improvement and she became very feverish. She then entered the hospital.

Physical Examination—A large corpulent woman, markedly prostrated, breathing very rapidly and looking desperately ill. Finger tips cyanosed. Face blanched and covered with perspiration. Tongue heavily coated. Chest—Percussion over anterior chest is resonant, except in the left axilla, where it is flat. Behind it, flat from the mid-scapular region down, on both sides. Throughout these areas there are showers of crepitant and subcrepitant râles. Inspiration is obscured by the râles. Expiration is softly bronchial, but differing in degree over different areas,—the consolidation seeming to be patchy.

Case No. 78551

Name M. W. Admitted August 27 Ward M. III





Heart—Outline cannot be made out by percussion. Action is exceedingly rapid but regular. No murmurs are heard. The pulse is small, poor force and rapid, low tension. Abdomen—The whole right side of the abdomen is very tender to pressure and slightly rigid, especially in the upper part, the signs being most marked just below the costal margin in the nipple line. Vaginal—Cervix dilated to admit tip of one finger. Slight lateral tear. No tenderness made out. Finger just reaches fundus, which seems to be firm. There is a moderate, foul smelling discharge. Leucocytes 27,000. Polynuclear leucocytes 85%, lymphocytes 15%. A diagnosis of septic endometritis and double septic pneumonia following abortion, was made.

A blood culture, taken the day of admission, was negative, but smears from the cervix showed numerous long chains of streptococci.

Ten c.c. of Aronson's antistreptococcus serum were injected daily from the 27th of August to the 7th of September with the exception of the 1st, when none was given, and of the 2d and the 30th of August, when two doses of 10 c.c. were given. Vaginal douches were given daily from August 27th until September 11th, and intrauterine douches were given from the 1st to the 11th of September. The return, at first cloudy and blood tinged, and containing a large amount of mucus with shreds of necrotic tissue, gradually became clearer, the necrotic shreds disappeared and the mucus decreased. During the early period cold sponges were given to reduce the temperature and colon irrigations of saline solution were given daily. The heart action was strengthened by strychnine sulphate, whiskey and digalen. The patient was irrational and thoroughly toxic.

At the end of the first week of treatment the symptoms had greatly improved. Tenderness which had been present over the uterus, was less marked, and the signs of consolidation in the lungs were diminished. The leucocytes count fell to 23,000 on the 4th of September and 14,000 on the 7th. On the 8th of September a marked edema of the face and hands occurred, with numerous urticarial wheals over the chest. This urticaria extended over the back and buttocks, accompanied by an erythematous eruption, which gradually extended over the entire body. This was accompanied by a leucocytosis of 23,000, which fell on the 13th to 9,800, accompanied by a fall of the temperature to normal. From this time on the recovery was uneventful with the exception of occasional pain in the right iliac region and occasional slight rises of temperature. The patient was discharged October 23d, cured.

REPORTS OF WARD CASES.

From the Services of
Dr. Austin W. Hollis and Dr. Theodore C. Janeway.

Otto H. Leber, M.D.

Brill's Disease.—It may not be out of place to report here a case of an acute disease, resembling typhoid, recently reported by Brill as a clinical entity, and of which he collected no less than 221 cases.

The patient was a man of 37, a Roumanian by birth. His past history is irrelevant except that he had an attack of typhoid at 14. Feeling perfectly well until 8 days before admission, he woke up one morning aching all over, and feeling very tired. Since then he has been ill in bed with a fever of 103-105 and suffering from general malaise and general body pains. There was no epistaxis, no chills, no vomiting, but anorexia, and constipation. For two days before admission there was a distressing cough.

On admission, patient appeared acutely prostrated, complaining greatly of general body aches and resenting being disturbed. The tongue was heavily coated, the throat congested, the tonsils enlarged. The heart was normal. A few râles were heard over the chest behind. The spleen could just be felt on deep inspiration.

Eruption: On the abdomen, chest, back, arms and upper part of thighs is a diffuse dull red, almost purplish maculo-papular eruption. The spots were very profuse and the majority did not disappear on pressure.

The patient's condition remained practically unchanged for 5 days more, when with a sudden break in the temperature from 105-102 there came a feeling of general well being and within 60 hours the temperature had reached 99, and the patient said he was feeling fine.

After coming to the hospital the rash spread over the entire body except the face, covering even the palms and soles of his feet and then gradually fading with the drop in temperature. Aside from the rash, which was entirely different in character and distribution from the ordinary rash seen in typhoid, the usual laboratory tests for typhoid (with the exception of one positive Diazo reaction) were entirely negative. Thus there were 2 sterile blood cultures and 3 negative Widal reactions and, of the daily blood counts, the lowest showed 14,000 white cells, the highest 21,000; while the differential count varied from 71-86% polymorphonuclears.

Brill's observations of cases of this disease date back 14 years; whether or not this is the first case of it that has occurred in the wards of this hospital is quite impossible to state, suffice it to say that a mere perusal of clinical notes or chart records can not possibly give the idea of clinical entity which one view of the patient, his eruption, and the daily return of negative laboratory facts gave to the observer and the conviction to him that here was a febrile disease, acute, self-limited, distinct, and not typhoid.

The case may be of further interest in demonstrating the successful use of phenolphthalein hypodermatically. The patient had been constipated practically all his life and had always resorted to cathartics of various kinds. One grain of diacetyl-phenolphthalein in suspension in olive oil was injected, besides which the patient was directed to drink water freely and include fruit in his diet. The patient has reported regularly since his discharge and though it is now only two months since the injection, the patient has had from one to three movements daily without once resorting to a cathartic.

In conclusion I beg to thank Dr. Hollis for the privilege of reporting this case and for his advice and suggestions in its preparation.

General Miliary Tuberculosis Fatal in Thirteen Days..—Case No. 82106. J. H. Schoolgirl; 19 years old. The patient was a well-nourished young girl, who had been exceptionally strong and active. Only one month before admission she had won first prize in a severe athletic contest.

The family history was negative; also her past history with the exception of occasional attacks of bronchitis.

The present illness began ten days before admission, quite suddenly, with nausea, headache, chill and vertigo. She had sharp, lancinating pain in the left side increased by deep breathing. There was some cough, and tenacious mucoid expectoration, which was slightly blood-tinged the day before admission. She had no sweats. She had had fever as high as 106, the first week, and had been so weak as to be in bed since the onset.

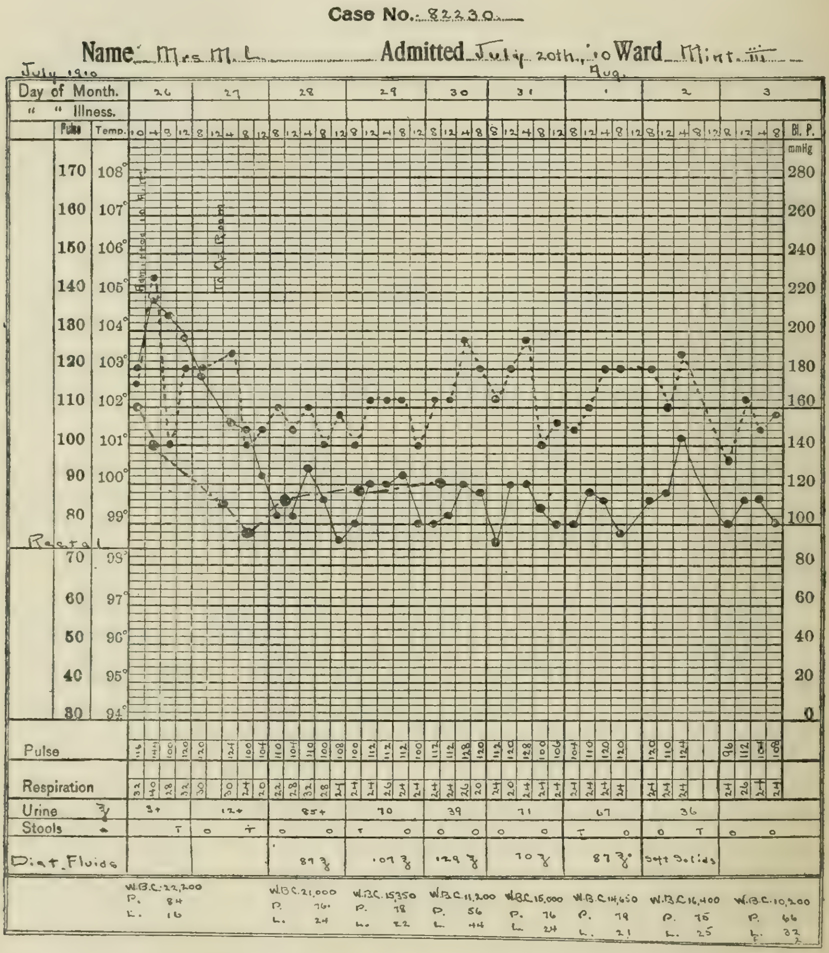
The physical examination on admission showed the girl to be markedly prostrated, with considerable dyspnea and marked cyanosis. The eyes (pupils and eye grounds), mouth and throat were negative. The neck was not rigid. The heart was normal. The lungs showed a very moderate dullness over the right upper lobe with slightly bronchovesicular breathing. The liver percussed two inches below the free border of the ribs and the edge of the spleen could be felt on deep inspiration. Extremities were normal. The clinical picture was peculiar in the great dyspnea and extreme cyanosis that became more marked day by day. Time and again the patient would start up in bed, gasping for breath and becoming livid for a few minutes, only to sink back again, exhausted, when temporary relief came in a few minutes. There was practically no change in her physical signs from day to day. The temperature ranged from 102-105, the respirations from 30-72, the pulse about 130. The blood count was 7,600 with 76% polymorphonuclears. The urine showed only a very faint trace of albumen with negative diazo test. Widal test and blood culture were negative.

A diagnosis of general miliary tuberculosis was made, and the patient died on the fourth day after admission and thirteen days after the onset. The diagnosis was confirmed by the finding of miliary tubercles in the spleen, liver and kidney, at a partial autopsy.

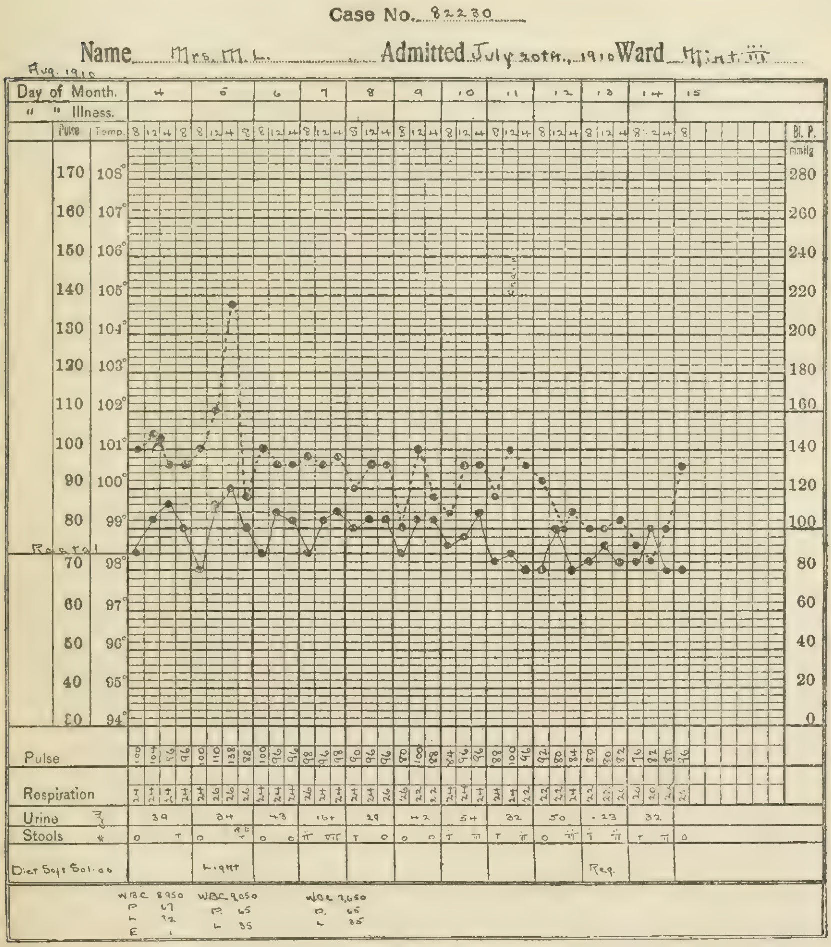
Puerperal Eclampsia Coming on One Week Past Partum.—Case No. 82,230. M. L. The patient was a young primipara of 26 with no history of previous illness. Her child was born one week before admission. The pregnancy had been complicated by frequent vomiting from the beginning of the fifth month but had become less frequent during the eighth month. The lower extremities had been quite edematous during the seventh and eighth month, and as this was becoming extreme, and serious kidney trouble was feared, the child was delivered with instruments at the end of the eighth month. After the birth of the child, the swelling subsided considerably; the baby, well and healthy, was nursed, and both mother and child progressed favorably for one week.

On the morning of admission, the patient suddenly complained of inability to see her child or those about her. Shortly afterward she went into a convulsion lasting about five minutes, and then had three others at intervals of about twenty minutes.

The patient came to the hospital in deep coma, with a temperature of 103, pulse 116, respirations of 52, and systolic blood pressure—160 mm. H g. She appeared well nourished, with no edema of the face and only very slight edema of both legs. The pupils were contracted equal, and reacted to light; the eye grounds were normal. The heart action was rapid, of good force, and there was marked gallop rhythm present. The lungs were negative. The hard firm fundus could easily be felt extending to just below the umbilicus. Knee-jerks could not be obtained. Babinski reflex was present.



The first day the patient had twelve convulsions, each lasting from three to ten minutes, and remained in coma the rest of the time. Under the administration of morphine, chloral per rectum, and colon irrigations, the patient became much quieter, but the next morning,



on account of the size of the uterus, and the continued unconsciousness, it was thought wise to curette.

A large quantity of friable and bloody tissue was removed, and the operation was attended by considerable hemorrhage. Scarcely two hours later the patient began to cry out occasionally, and ask for water. After this, recovery was rapid and uneventful. She refused at first to believe she had a baby, but its presence soon brought back her memory. The temperature rapidly fell to normal. The urine, which at first was scanty and loaded with albumen, became abundant on the following day, and the albumen rapidly diminished to a very faint trace. The blood pressure decreased from 160 to 110-120. Today, about three months after her attack, she feels perfectly well.

In conclusion, I beg to thank Dr. T. C. Janeway for the privilege of reporting these two cases from his service and for his kind advice and suggestions in their preparation.

Children's Service

CHILDREN'S SERVICE FOR 1910

DISEASES DUE TO MICRO-ORGANISMS INFECTIVE DISEASES	Sex		Results				
	Male	Female	Cured	Improved	Unimproved	Died	Totals
Diphtheria	...	3	3	...	...	...	3
Diphtheria, incontinence of urine	...	1	...	1	...	...	1
Diphtheria (nasal), hare lip	1	...	1	...	...	...	1
Gonococcus vaginitis, catarrhal jaundice	...	1	...	...	1	...	1
Lymphatic fever	1	...	...	...	...	1	1
Malaria	1	...	1	...	...	...	1
Malaria (tertian)	1	1	2	...	...	...	2
Measles	1	...	1	...	...	...	1
Pertussis	1	...	...	...	1	...	1
Rheumatism (ac. articular)	1	...	1	...	...	...	1
Rheumatism (ac. art.), mitral stenosis and insufficiency	2	3	...	5	...	...	5
Syphilis (congenital)	...	1	1	...	...	...	1
Syphilis (congen.), gastro-enteritis	...	1	...	1	...	...	1
Syphilis (congen.), interstitial keratitis	...	1	...	1	...	...	1
Tuberculosis of hip, ac. articular rheumatism	...	1	...	1	...	...	1
Tbc. of spine	1	...	...	1	...	...	1
Tbc. meningitis	3	5	...	...	2	6	8
Tuberculosis (miliary), tbc. meningitis	1	...	...	...	...	1	1
Typhoid fever	6	1	7	...	...	...	7
Typhoid fever with relapse	1	...	1	...	...	...	1
	21	19	18	10	4	8	40
ALIMENTARY SYSTEM INTESTINES							
Colitis	1	1	1	1	...	...	2
Constipation	...	1	1	...	...	...	1
Dysentery	1	...	1	...	...	...	1
Gastro-duodenitis	...	1	1	...	...	...	1
Gastro-enteritis	12	7	15	...	1	3	19
Gastro-enteritis, malnutrition	...	2	...	...	...	2	2
Ileo-colitis	5	2	4	...	...	3	7
Ileo-colitis, malnutrition	3	1	...	...	...	4	4
Ileus, general peritonitis	...	1	...	...	1	...	1
	22	16	23	1	2	12	38
PHARYNX							
Hypertrophy of tonsils, adenoids, cervical lymphadenitis	1	...	...	1	...	...	1
RECTUM							
Stricture of anus, recto-vaginal fistula, chr. constipation	...	1	...	1	...	...	1
STOMACH							
Gastritis (acute)	1	...	1	...	...	...	1
Gastric tetany	...	1	1	...	...	...	1
	1	1	2	...	...	...	2
CARDIO-VASCULAR SYSTEM BLOOD							
Hemophilia neonatorum	1	...	...	...	...	1	1
Splenic anemia	1	...	...	...	1	...	1
	2	...	...	...	1	1	2

	Male	F.	C.	Imp.	Un.	D.	Tot.
HEART							
Dilatation, mitral insufficiency	2	2	...	1	...	3	4
Pericarditis (acute), mitral insufficiency	...	1	...	1	...	...	1
Valvular disease—mitral insufficiency	1	3	...	4	...	...	4
	3	6	...	6	...	3	9
DUCTLESS GLANDS							
Hyperthyroidism	...	1	1	...	...	...	1
Myxoedema (infantile)	...	1	...	1	...	...	1
	...	2	1	1	...	...	2
MUSCULAR SYSTEM							
Muscular atrophy	1	...	...	...	1	...	1
NERVOUS SYSTEM							
DISEASES OF THE MIND							
Idiocy	1	...	...	...	...	1	1
NERVOUS DISEASES OF UNKNOWN ORIGIN							
Chorea	2	2	2	2	...	...	4
Chorea, chr. endocarditis	3	2	...	5	...	...	5
Convulsions, cerebral hypersusceptibility	...	1	1	...	...	...	1
Convulsions of intestinal origin	1	...	1	...	...	...	1
Convulsions of unknown cause	...	2	1	1	...	...	2
Spasm (habit)	...	1	...	...	1	...	1
	6	8	5	8	1	...	14
SPINAL CORD							
Poliomyelitis (acute anterior)	3	1	...	2	2	...	4
Sclerosis (multiple)	...	1	...	...	1	...	1
	3	2	...	2	3	...	5
OSSEOUS SYSTEM							
BONES							
Rickets	2	1	...	3	...	...	3
Rickets, malnutrition	...	1	...	1	...	...	1
	2	2	...	4	...	...	4
RESPIRATORY SYSTEM							
BRONCHI							
Bronchitis	5	4	8	...	...	1	9
Bronchitis (acute), adenoids, mitral insufficiency	...	1	...	1	...	...	1
Bronchitis (ac.), axillary adenitis	1	...	1	...	...	...	1
Bronchitis (ac.), diphtheria, mitral insufficiency	...	1	...	1	...	...	1
Bronchitis (ac.), eczema	1	...	1	...	...	...	1
Bronchitis (ac.), gastro-enteritis	1	...	...	1	...	...	1
Bronchitis (ac.), malnutrition	...	1	1	...	...	...	1
Bronchitis (ac.), rachitic deformities	...	1	...	1	...	...	1
Croup	...	1	1	...	...	...	1
	8	9	12	4	...	1	17
LUNGS							
Pneumonia (broncho—)	6	2	6	...	...	2	8
Pneumonia (broncho—), gastro-enteritis	...	1	1	...	...	...	1
Pneumonia (broncho—), ileo-colitis	1	1	1	...	...	1	2
Pneumonia (broncho—), inanition	...	2	...	...	...	2	2
Pneumonia (broncho—, terminal), malnutrition	...	1	...	...	...	1	1
Pneumonia (lobar)	6	9	12	...	...	3	15
Pneumonia (lobar), hypertrophied tonsils and adenoids	2	...	2	...	...	...	2
Pneumonia (lobar), ileo-colitis	1	...	1	...	...	...	1
Pneumonia (lobar), malnutrition	...	2	...	...	...	2	2
Pneumonia (lobar), spastic diplegia	1	...	...	...	1	...	1
Pneumonia (lobar), suppurative pleurisy	2	2	...	3	1	...	4
Pneumonia (unresolved), congenital cardiac disease, pertussis	...	1	...	...	1	...	1
	19	21	23	3	3	11	40

	Male	F.	C.	Imp.	Un.	D.	Tot.
PLEURAE							
Empyema thoracica	1	...	...	1	...	...	1
Pleurisy with effusion	2	...	2	...	...	...	2
Pleurisy with effusion, otitis media, mastoiditis	1	...	...	1	...	...	1
	4	...	2	2	...	...	4
ORGANS OF SENSE							
ORGAN OF HEARING							
Otitis media, malnutrition	2	1	3	1	...	...	3
ORGAN OF VISION							
Blepharitis, abscess of hand	1	...	1	...	...	...	1
Optic atrophy due to hydrocephalus	1	...	...	...	1	...	1
	2	...	1	...	1	...	2
TEGUMENTARY SYSTEM							
Eczema	1	2	3	...	...	...	3
Furunculosis	1	...	1	...	...	...	1
Miliaria Papulosa	...	1	...	1	...	...	1
	2	3	4	1	...	...	5
URINARY SYSTEM							
KIDNEY							
Nephritis (acute), pyelitis	1	...	...	...	1	...	1
Nephritis (post-scarlatinal)	...	1	1	...	...	...	1
Nephritis (sub-acute)	...	1	...	1	...	...	1
Pyelitis, cystitis	...	1	...	1	...	...	1
	1	3	1	2	1	...	4
CONGENITAL MALFORMATIONS							
Cong. cardiac disease, malnutrition	...	1	...	...	1	...	1
Cong. hare lip and cleft palate	...	1	...	...	1	...	1
Cong. hydrocephalus, lobar pneumonia	...	1	...	...	1	...	1
Cong. pyloric stenosis, general peritonitis	1	...	...	...	...	1	1
	1	3	...	...	3	1	4
NEOPLASMS							
Fibro-sarcoma of nose	...	1	1	...	...	...	1
Tumor of knee	1	...	...	...	1	...	1
	1	1	1	...	1	...	2
INTOXICATIONS AND POISONS							
Auto-intoxication	1	...	...	...	...	1	1
MISCELLANEOUS CONDITIONS							
Boarder	4	3	5	1	1	...	7
Feeding case	2	1	3	...	...	...	3
Inanition	1	1	...	...	...	2	2
Inanition due to prematurity	...	1	...	...	...	1	1
Malnutrition	13	14	21	4	1	1	27
Marasmus	4	5	1	...	...	8	9
Marasmus, gastro-enteritis	2	...	...	...	...	2	2
Marasmus, sub-cutaneous abscesses	...	1	...	...	...	1	1
No pathological condition	1	1	...	...	2	...	2
	27	27	30	5	4	15	54

A SPORADIC CASE OF EPIDEMIC LYMPHATIC FEVER.

Chas. F. Collins, M.D.

Recently there was admitted to the Children's Medical Ward a case of lymphatic fever, and as it followed the classical outline so closely it seemed to me of enough interest to record; in addition there was an unusual feature, and that was its extreme duration; the greater number of cases run their course, as a rule about three weeks, whereas in this particular instance the length of time was considerably extended.

Epidemics of lymphatic fever have been reported from time to time, isolated cases are often not recognized, and as the disease is not as a rule described in general text books and not often met with, it may not be out of place to give it a short picture based on some of the published reports of epidemics and a small outbreak I observed in 1906.

The disease is spoken of as glandular or lymphatic fever, yet the lymphatics in the true sense of the word are not glands, and many spurious or false cases are cited, which are simply enlargements of the nodes of the neck or other portions of the body due to local infection, and these do not have any strict bearing or similarity to the true epidemic of lymphatic fever.

The disease was not known and recognized as a special one until 1889, when it was first discovered by Dr. Pfeiffer, of Wiesbaden, who described an epidemic under his care. The epidemic lymphatic fever is an acute infectious disease characterized by sudden onset with enlargement of the lymphatic nodes and channels, beginning always in the neck, extending throughout the whole chain, except possibly the lymphatics of the extreme extremities, and also enlargement of the liver and spleen as a rule. It is not fatal; I find the record of only one death, and that of a very feeble child just recovering from a severe illness. So far as I know the disease occurs only in childhood. In the Wiesbaden epidemic most of the cases were between five and eight years of age, the oldest being sixteen. In the outbreak under my observation, I had one infant eleven months of age. Even younger were recorded. The period of incubation is estimated at seven days.

The onset is fairly acute, usually some headache, malaise and a pharyngeal cough, considerable prostration, with a rise of temperature two or three degrees. In one of my cases the sudden onset was accompanied by a temperature of 103.8° in a child of six and one-half years of age. There is often vomiting and loss of appetite. The child complains of pain in the throat, more particularly the pain in the neck as a whole, especially on motion, and on making the effort to swallow.

The picture is one of a child suffering from a profound infection. The mucous membrane of the throat is red, the tonsils may be swollen, and there may be patches of membrane, sometimes a coryza. By the first day, or certainly the third, one can easily feel an enlargement of the lymphatics of the neck on both sides extending as a chain down to the clavical. The nodes are not changed in outline, but their enlargement may be considerable, are very tender on palpation, but firm and on rotation of the head with motion of the neck painful, do not show any fluctuation and do not break down. By degrees the enlargement of the lymphatics extends downwards into the trunk. The superficial axillary and clavical lymphatics may be easily felt, also the inguinal, and those of Scarpa's triangle. The epitrochlear I noticed in one case, but have never observed any in the popliteal space. It has been noted by some that this advancement of the enlargement of the lymphatics would be more in advance on one side than on the other. One author claims the right. It was so in one of my cases.

The mesentery lymphatics are enlarged and on one occasion I could feel them distinctly through the thin abdominal wall. The mediastinal and those near the oesophagus are in all probability involved. There is a sharp pain about the umbilicus, especially midway between the umbilicus and the ensiform. This has been especially claimed as a characteristic symptom. There is pain over the lower portion of the sternum, at times spontaneous, and on pressure, and there is pain over the sternum during the act of swallowing. The liver and spleen are usually enlarged and somewhat tender. In this sporadic case the liver was 2½″ below the free border of the ribs, and the spleen 3″, and this at the time of convalescence. There is no oedema of the extremities, but movement causes pain in the region of the groin or axilla as the case may be. The lungs and heart give no evidence of involvement; enlarged bronchial nodes are not made out by percussion.

The development of this picture consumes six to eight days. The constitutional symptoms then abate and the physical conditions gradually return to normal. In the case (the subject of this report under observation three months), the liver and spleen were still enlarged, though much diminished since first seen, but the lymphatic enlargements have almost disappeared. In general one expects acute constitutional symptoms to improve by the end of the week; by 21 days, possibly four weeks, the examination is usually negative. Very mild cases, of course, are shorter.

In the earlier epidemics I find no record of blood counts or throat cultures. I am sorry I cannot give a full blood picture, but unfortunately in all of the cases I saw in 1906 observations were not made. As a sample though, one case 48 hours after the onset showed 14,800 leucocytes, polynuclears 62½, small mononuclears 32, large mononuclears in transitional state 5.4, the eosinophiles 5%. On the eighth day of the disease the leucocytes were 10,000, the differential count normal; that child was eleven months of age.

In a case 6½ years of age on the third day there were 19,000 leucocytes, polynuclears 70%, lymphocytes 24%, l. mononuclear 5%, eosinophiles 1%; on the 9th day 7,600, and differential normal. In the above mentioned sporadic protracted case, two weeks after onset there was some rise of temperature, the whites were 12,500, the reds 5,000,000, the hemoglobin 87%, polys 43%, lymphocytes 57%. Eight days later, when the temperature had been normal for the same length of time, the white cells were 10,000, the polys 59%, the lymphocytes 41%. The child's age was 7½ years.

Such throat smears as I have records of showed staphylococci and streptococci, and in one case an unclassified bacillus was noted. I would here mention that I have never seen a parotid gland enlarged and I find no record of the same.

This is a general composite picture of the cases coming under my observation.

The type and picture correspond very closely to the first epidemic recorded by Pfeiffer in Wiesbaden in 1889, and also to the rather extensive epidemic in Ohio in 1896 recorded in the most careful and complete manner by Dr. J. Park West, which report he read before the Academy of Medicine. He recorded 96 cases arising in 43 families in the town of Businessburg, O., and cases in the adjoining county. This epidemic was protracted during the cold months of '94 and '95 and into the spring of '97.

Pfeiffer, in his Wiesbaden epidemic, mentioned the mild, short form, and said if these cases had not arisen in families where other children had the severe type, he would have been at a loss to diagnosticate them, and certainly to classify them. In mild cases there is a painful throat, pain on swallowing, and pain in the neck, especially on motion. The neck lymphatics are enlarged, and in some cases the posterior ones more than the anterior. There was moderate temperature, little malaise, no prostration, patients were apt to be up and about, and in two or three days recovered. He also mentioned some of his cases having coryza symptoms. These I have not seen.

Through the literature many cases of so-called cases of glandular fever are recorded, most of these are enlargement of lymph nodes accompanied by fever, the swelling subsiding gradually, or soften and form abscesses, and as a rule are referable to various neighboring infections, or some constitutional condition. These I feel must not be classified under the head of lymphatic fever. Using the word "glandular fever" as a synonym for lymphatic fever causes this confusion.

With the short foregoing picture of the disease, I would here record the case of C. L. 7½ years of age, which was sent to my service in August, 1910, by Dr. F. S. Meara, and the case, after being discharged from the hospital, was seen at intervals as late as January, 1911.

Chief Complaint—Cough, fever, pain in chest.

Present Illness—About two months ago had slight cough and mother noticed that she was somewhat feverish. No diagnosis had been reached, although tuberculin and Widal tests and urine and blood examinations have been made. She has been sick in bed during the past ten days, and mother has taken her temperature. Temperature has usually been normal in morning and has risen in evening as high as 103° and 105° a couple of nights during past few days. Appetite has always been poor even when well, but has been worse lately. Bowels are regular. Cough has been very severe. No expectoration. Has always passed urine frequently and has sometimes incontinence at night. Mother has never noticed anything peculiar about urine. During her illness she has often complained of pain at lower end of sternum, made worse by swallowing. Has complained of headaches during past week.

Past History—Had measles when 2 years old, No other sickness except intestinal disturbance when an infant.

Family History—Father and mother well; three other children in family, all well.

Patient is a well nourished child of 7½ years, who looks moderately ill. Skin is clear, mucous membranes rather pale. Eyes and ears appear normal. Tongue is clear, pharynx is normal. Tonsils not enlarged. Teeth in fair condition, except for two or three cavities in molars. Lymph nodes show enlargement as follows:—Right post cervical chain—one node about 1 cm. in diameter and several smaller ones. Left post cervical chain—several small nodes. Right ant. cervical—one node at angle of jaw about 1 cm. Several smaller nodes, diminishing in size from above downwards. Left ant. cervical—Two or three small nodes at angle of jaw. Right axillary—two or three small nodes. Left axillary—one large node. Right epitrochlear—one small node. Left epitrochlear—none felt. Inguinal chains—numerous nodes varying in size, also in lower part of chain in scarpa's triangle. Chest—well developed. Lungs—appear clear. Heart—apex in fifth space 2½ inches from mid line. Sounds of good quality, action regular, no murmurs. No enlargement to right. Abdomen—edge of liver felt 2½ inches below costal margin. Spleen—lower pole extends 3 inches below costal margin, does not extend far towards median line, but appears rather long and slender displaced downward. Extremities—knee jerks present; no oedema nor paralysis. Spine—appears normal; no dullness on percussion over dorsal region.

NOTES—Aug. 24. 1910. Vaginal smear neg. for gonococci. W. B. C. 12, 500 P 43 L57 12N. Very quiet night. Coughs frequently.

Aug. 25. Vaginal smear neg. for gonococci. Urine, catheterized specimen, neutral turbid, 1022, albumen O, sugar O. Micros:—Few leucocytes, epith. cells, phosphate M sediment.

Aug. 26. R. B. C. 5,000. Hgb 78%. Coughs very little now.

Aug. 27. Patient still complains of slight headache and pain at lower end of sternum on swallowing after eating.

Aug. 29. Throat Culture (25th) showed staphylococci.

Aug. 30. Patient's serum gives a negative Wasserman reaction. Patient feels better. Lymph nodes in neck have decreased slightly in size.

Sept. 1. W. B. C. 10.900. P 59 L 41.

Sept. 2. Liver and spleen seem to have diminished slightly in size.

Sept. 4. Patient still complains of slight headache at times, but eats and sleeps well.

Sept. 5. Allowed up in chair.

Sept. 7. Lymph nodes in both anterior and posterior cervical regions have decreased considerably in size, also those in inguinal regions. Glands in Scarpa's triangle do not seem any smaller. Edge of liver descends 2 inches below costal margin in nipple line. Lower pole of spleen felt 2¼ inches below costal margin on deep inspiration. Patient eats and sleeps well. Still complains of pain at lower end of sternum after eating, but this does not seem so marked; has gained considerable weight and looks well.

Sept. 10. Vaginal smears negative for gonococci.

Sept. 12. General condition good. Spleen felt 2 inches below. Liver edge, 3 inches below costal margin. Lymph nodes in left axilla are large. Cervical nodes smaller. Heart—action slightly irregular. Second pulmonic accentuated. Soft systolic at pulmonic area. Fainter systolic at apex. Abdomen—circumference at umbilicus 20¾ inches. Still complains of pain at lower end of sternum for a short time after eating.

The temperature chart is not given, as after five days it fell from 102° in the morning to normal, and did not rise higher than 99°, and on two occasions fell to 96° at four in the afternoon. A urine daily record was kept, which was without clinical significance, except slight increase in frequency the first few days after admission. On Sept. 14, the patient's general condition was good and the child was allowed to go home, the mother being requested to bring the child to the hospital from time to time for further observation, which plan was carried out.

It is to be regretted that observations could not be made in detail during the first two weeks the child was confined to bed, yet the picture given by the mother corresponded very closely to the picture of the disease as given in the preliminary remarks.

The child came to the hospital on several occasions for examinations, and on each occasion a diminution in the size of nodes, as well as spleen and liver, was noted. On Jan. 28, 1911, the child was examined at her home. The right posterior cervical nodes were very small—about the size of a pea. The right inguinal nodes were moderately enlarged, not tender; the left inguinal a little larger than normal. The spleen edge felt just under free edge of ribs; the liver edge not made out. The child was well nourished, somewhat pale, appetite poor. The mother considered the child in very good health.

The history of this case, as mentioned at the outset, is of interest on account of the length of time, not only during the acute phase, twenty-five days, but its subsequent run and observation for 18 weeks longer, at the end of which time the inguinal nodes were the only significant physical abnormality.

As to etiology, nothing of importance can be said. Theories have been suggested, mostly pointing to infection through naso-pharyngeal region, some suggesting an intestinal origin, but the question remains to be solved at some future time.

References to some of the literature which has been of value in presenting this case are here added:

Roussel, Philadelphia Med. Bulletin, Vol. XIX, p. 131.
West, Archiv, Pediatric, Vol. XIII, 1896.
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Hesse, " " Vol. XLII, 1896.
Hainebach, Deut. Med, Wochenschrift XXV.
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Orthopedic Service

ORTHOPEDIC SERVICE

DISEASES OF THE SPINAL CORD	Cured	Improved	Unimproved	Died	Totals
Paraplegia (spastic)	...	2	...	...	2
Poliomyelitis (anterior)	...	4	...	...	4
Poliomyelitis (ant.) paralysis	...	4	...	...	4
Poliomyelitis (ant.), paralysis, talipes equinus	...	1	...	...	1
	...	11	...	...	11
DISEASES OF THE BONES					
Chondrodystrophy	...	...	1	...	1
Rickets (acute)	...	1	...	...	1
	...	1	1	...	2
DISEASES OF THE JOINTS					
Arthritis of knee, paralysis of quadriceps extensor muscle	...	1	...	...	1
CONGENITAL MALFORMATIONS					
Absence of tibia	...	1	...	...	1
Dislocation of hip	...	5	2	...	7
Malformation of femur, talipes equino varus	...	1	...	...	1
Malformation of frenum	...	1	...	...	1
Malformation of lumbar-sacral vertebrae, spondylolisthesis, knock-knees	...	1	...	...	1
Spondylolisthesis	...	1	...	...	1
	...	10	2	...	12
DEFORMITIES					
Contraction of knee from septic arthritis	...	1	...	...	1
Genu varum	2	...	...	...	2
Pes planus	2	1	...	...	3
Talipes equino varus	1	1	...	...	2
Talipes equino valgus, spastic paraplegia	...	1	...	...	1
	5	4	...	...	9
DISEASES DUE TO MICRO-ORGANISMS					
Syphilis (hereditary)	1	1	...	...	2
Syphilitic osteitis of knee	...	1	...	...	1
Tuberculous arthritis of ankle	...	1	...	...	1
Tuberculous arthritis of hip	...	3	...	...	3
Tuberculous arthritis of knee	...	1	...	...	1
Tbc. of hip with abscesses	...	1	...	...	1
Tbc. osteitis of knee	...	1	...	...	1
Tbc. osteitis of spine	1	3	...	...	4
Tbc. osteitis of spine and hips, perforation of colon	...	...	...	1	1
Tbc. osteitis of spine, hips, and both knees	...	1	...	...	1
	2	13	...	1	16
MISCELLANEOUS					
Marasmus	...	...	1	...	1

SOME GENERAL CONSIDERATIONS IN THE TREATMENT OF INFANTILE PARALYSIS.

T. Halsted Myers, M.D.

The very great increase in the number of cases of Poliomyelitis, within the last three years especially, has thoroughly alarmed the profession and the public as well. It is not my intention to discuss the many valuable investigations which have resulted from this widespread interest. The practical results have been that it is universally regarded now as an infectious disease, though the organism has not been demonstrated as yet. Neither has any antitoxin, or serum treatment or method of vaccination been elaborated sufficiently to warrant its use on man. That we are on the eve of such a discovery seems probable. Meanwhile the state of New York, as well as many other states and many municipalities, has recommended the same quarantine precautions for cases of this disease as are used against other communicable diseases. The New York Board of Health include under "Communicable Diseases," Diphtheria, Typhoid Fever, Tuberculosis, Epidemic Cerebro-Spinal Meningitis, Whooping Cough, etc.

Dr. Eugene H. Porter, State Commissioner of Health, has answered a letter of mine on this subject as follows:

"The most contagious period of epidemic poliomyelitis is supposed to be its catarrhal or acute stage. Just how long it is possible to transmit this disease has not as yet been definitely established. The duration of quarantine as recommended by this Department is four weeks. I have also recommended that thorough disinfection of the stools and urine should be insisted upon, and on the completion of the case the room or apartment in which the patient has been should undergo a thorough course of disinfection. While we do not as yet fully understand the disease, its severity is such that we should enforce none but the strictest measures to ensure protection to others."

At the Rockefeller Institute, as Dr. Cole very kindly informs me, the plan has been to isolate fairly strictly the cases during the first six weeks, but the mothers have been allowed to visit them. In cases of longer duration, eight weeks, the children were kept in separate rooms but were not isolated strictly. He considered the most important point in preventing the spread of the disease was careful attention to the nasal secretions and possibly also to any mucus expectorated. All these secretions should be burned. The period of infectivity may be less than that mentioned, but the whole matter is as yet obscure. The walls, floor and furniture of the room is afterwards cleaned with a disinfectant solution, and the bed sterilized. Experiments conducted so far with the virus seem to show that formaline has little or no effect.

The period of incubation varies considerably, but is generally from one to two weeks in the epidemic cases. The sporadic cases, and cases developing, one, two or three months after an epidemic in the same locality, have led to the supposition of a very slow incubation in some instances. The length of time that quarantine should be continued is not certain as yet, but legally it is four weeks.

It would appear wise to screen sufferers, in the acute stage of the disease at least, from flies, mosquitoes, etc., as possible germ carriers. As the nose and throat are now regarded with especial suspicion as ports of entry of the infectious organism, any abrasion or inflammation of the mucous membrane in this location should receive prompt and continuous treatment in the patient and attendants. It would seem rational moreover, also to take antiseptic precautions whenever other mucous membranes or the skin are injured or inflamed.

Clinically, the onset of poliomyelitis varies greatly. In some cases there are no prodromal symptoms at all. For many years I have been greatly interested in this disease as most of these cases come sooner or later to the Orthopedic surgeon. I have therefore had many cases whose parents were very observant, also many cases who were carefully observed by competent physicians from the first, and they have been subjected to the most rigid examination as to the condition of the child prior to, and during the acute stage; specifically, inquiry was, as a routine method, made as to malaise, fever, constipation, diarrhoea, vomiting, cough, coryza, and for the past two years abrasions of the skin and insect bites have been asked about. One often sees in articles on this subject some general remark to the effect that where prodromal symptoms were not found they had been overlooked. Such cases are not common, but my contention is that they are not rare either.

It is very difficult in this disease, as indeed in any other disease, to estimate the value of treatment. We can relieve any symptoms, but how much this will influence the course of the disease is another question. The onset in two cases may be identical as far as we can judge, yet the resulting paralysis may vary greatly in distribution and degree. Why is this? Has one individual more power of resistance to the effects of the infection, or has the other a greater or more virulent infection? We believe both these suppositions may be true, but what I wish to call attention to, is that we have no certain gauge for these conditions.

Treatment therefore in the acute stage, until we receive from the laboratory the hoped for specific, must be symptomatic. In addition to this on general principles active catharsis is indicated at once to reduce a possible increase of the auto-intoxication from the intestinal tract, and to lower the blood pressure, since the very great rapidity of the onset of the paralysis suggests a gross mechanical element in the exciting cause in addition to the organism. So many cases are attacked almost immediately after a sudden chilling of the body surface, as after a cold plunge, or after sitting on the cold ground, while overheated: in what manner the susceptibility of the person is thus increased is a matter of conjecture only; perhaps a sudden congestion of the spinal cord occurs.

We cannot distinguish the sporadic cases pathologically, or in their clinical results from those occurring in epidemics. In the latter cases it seems established that the damage to the nerve centers is effected simultaneously with very rapid changes in the character of the spinal fluid, as determined by repeated lumbar punctures before, during and after the onset of the paralysis.

As on microscopic examination there is found congestion of vessels in the anterior horns of the cord, perivascular infiltration, hemorrhages etc., in the acute stage, it would seem proper to try to relieve congestion and tension. We cannot prove that this can be done, but anyone who is familiar with the essay of Hood on "Rest and Pain" cannot but be impressed with the marked influence exerted on the nerve centers, clinically, by treatment of the superficial nerves from these centers.

Counter irritation of the skin over the area of the cord involved might therefore be expected to relieve the spinal congestion. This can be best done by mustard applications, repeated often, care being taken that the epidermis is not injured. Where pain is a marked symptom gentle friction in a warm bath often gives marked relief.

When we come to the consideration of the treatment of the paralysis, we must remember that any muscle, however strong and healthy, can be tired out easily by over use, and to these damaged muscles, any use is over use, and so definitely injurious.

Absolute rest in bed is indicated at first, whether there is pain or not; and this should be continued much longer, I believe, than is usually done. It is the natural wish of the child, and the parents, to see how much power there is. To continually test this in various ways, they urge the child to move, to stand, to walk, etc., at great risk of exhausting the weakened muscles. Then, too much treatment is usually insisted upon: massage, manipulations, the various forms of electricity; to make sure that everything possible is being done for the patient. All this is wrong, I believe, at least for the first two, and probably four, weeks. The affected parts should be kept warm, wrapped in wool or cotton, and heat applied externally also, if necessary, but nothing more.

Furthermore, the distribution of the paralysis varies greatly, not only in the number of the limbs affected, but in the completeness of the paralysis in the individual muscles. There is no way at the onset of the disease to determine how extensive the ultimate disability will be. In the cases I have seen during the last three or four years this has been especially true. Not only have I seen what may be called abortive cases, cases which presented the usual prodromal symptoms but did not become paralyzed, and others showing a pronounced and sometimes extensive muscular weakness which disappeared in a few days; but even in the cases with extensive and apparently quite complete paralysis, the recovery has been very much more complete than I should have expected, judging from former experience. Another difference I have noted in these recent cases is that a greater proportion of them have sensory symptoms, pain in the affected parts for the first few days, or for a week or two.

At the end of two weeks a careful examination should be made of the amount of voluntary motion possible. Is it possible to flex or extend the toes, ankle, knee, hip, fingers, elbows, shoulder, etc.? These results should be recorded. From this examination we can determine what deformity is threatened. We know that less paralyzed muscles will overpower the weaker ones, and gradually become contracted, unless the force of gravity, or our treatment, prevents this. When all muscles are paralyzed, gravity will determine the deformity, and contractions of ligaments or fasciae will perpetuate it; but all the muscles are seldom paralyzed; whole groups are sometimes affected, but generally only individual muscles in these groups. This is sufficient to destroy the balance of power however, and almost invariably some distortion is to be expected. For example: paralysis, even though

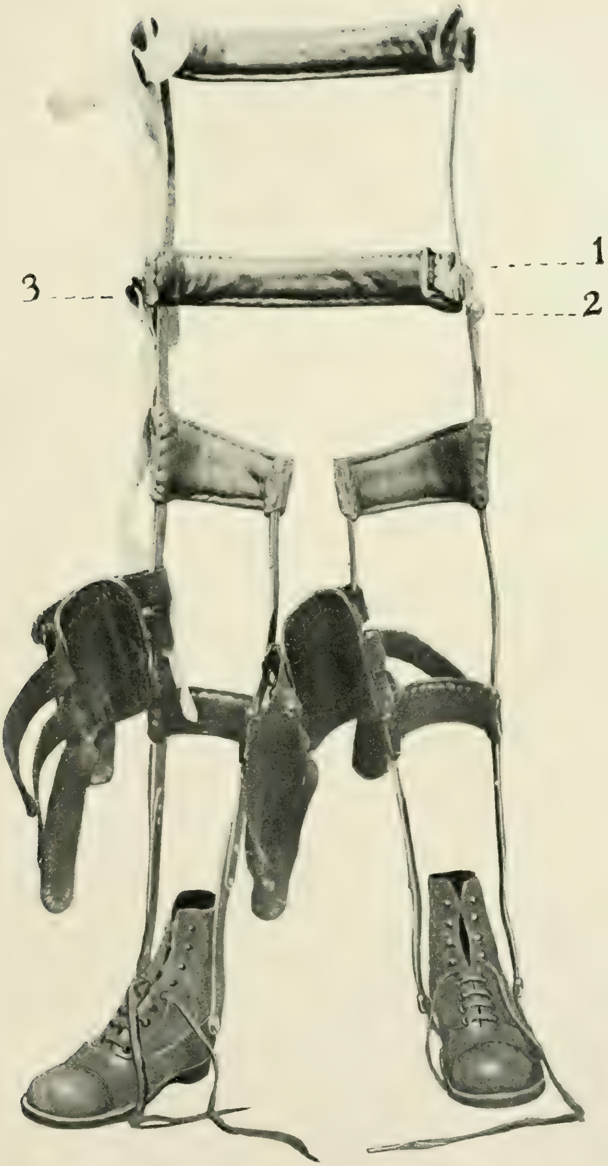

Fig. 1.—(1) Joint allowing flexion and extension; (2) Hinge allowing abduction and adduction; (3) Snap joint, holding hip rigid, or allowing flexion, at will.

partial, of the anterior tibial muscle alone, will impair dorsal flexion and adduction at the ankle enough to produce equinus and valgus: or a weakened peroneus longus alone, will cause a varus deformity.

Until the patient is allowed to walk, it is not necessary to treat this muscular inequality, unless the stronger muscles are found to be contracting. In that case the condition must be treated at once, even before walking is permissible, for the double purpose of avoiding the deformity itself, and of preventing undue strain on the weakened opposing muscle, whose integrity it is most important to preserve.

The appearance of these contractions must be watched for, and opposed, as soon as they are found. It occurs in some cases much sooner than in others, and is due not only to the degree of the difference of power in the opposing groups of muscles, but also to an apparent increase in the normal tonicity of the muscles least paralyzed, or not paralyzed at all. In these cases also, efforts to overcome the contractions cause more pain than in cases where the deformity occurs more slowly.

If for example we find that dorsal ankle flexion is possible only to 85°, or 90°, instead of the normal 70°, it is time to apply an ankle brace which will keep the foot flexed at 85°, and to begin stimulation of the weaker muscles. For this purpose the most valuable means I have found to be massage, heat, and vibration. The value of electrical stimulation I think is greatly overestimated; it has no value except as a method of exercise, and here it has its place. The amount and character of the current necessary to produce a contraction is also a convenient method of measuring the recovery of the affected muscle. Electrical examinations as a rule are however extremely unsatisfactory for making a prognosis, as not one man in ninety can make a reliable examination in a child in this class of cases.

Although an almost unlimited variety of deformities may be caused by these paralyses, the most common are, at the ankle, equinus, or calcaneus, with or without varus or valgus: at the knee, flexion; at the hip, flexion with abduction, from contraction of the tensor vaginae femoris; at the shoulder the pectoral muscles most often escape and become contracted: at the elbow no deformity occurs; at the wrist, palmar flexion, adduction of the thumb, and extension of the fingers also occasionally lost. The most severe and intractable cases of scoliosis are caused by paralysis, unequal on the two sides, of the short and deepest layers probably, of the spinal muscles.

The main objects of treatment are, to protect the weakened muscles and to stimulate their vitality; to prevent or relieve deformity, and insure growth as near normal as possible; to increase manual power, or locomotion, by means of splints, muscle transference, or nerve anastomosis : and finally, in these ways indirectly to benefit the general health, and especially the mental complexion of these distressing cases.

I have spoken several times already of the necessity of protecting the weakened muscles, as I think it is the most important means of assistance we have, especially in the first year of treatment. This is done by preventing or overcoming the contractions in the stronger opposing muscles, and by supplementing their own power by braces or operative means. For instance, when equinus is developed, we stretch mechanically, or elongate surgically, the tendo-Achillis, so that ankle flexion is possible to about 80° passively; we then also relieve the anterior muscles from the strain of the weight of the fore-foot by an ankle brace. This brace must be made as light as possible, and have a stop joint which will allow the foot plate to extend only to 90°, but to flex freely. By raising the inside of the foot plate, or the outer side, we can also prevent valgus or varus deformity at will.

Surgical interference, where the patients can be watched, should in nearly all cases be deferred at least two or three years. Where the patients are not watched carefully, contractions may occur early, and become fixed, long before this time, and in this case it will be necessary to straighten the feet, knees, and hips, before braces can be applied to the best advantage. Often, some recovery of power is noticed at the end of a week or two; generally, a considerable increase in power is seen in two or three months. This improvement is most marked in the first year, less in the second, and still less in the third: about the age of puberty, in a number of my cases, there has seemed to occur another period of improvement in the paralyses affecting the lower limbs.

The function of the affected joint depends not only upon proper support being afforded to it, but also upon the condition of the associated joints.

If the quadriceps extensor at the knee is paralyzed permanently we consider three methods of relief; holding the knee stiff during locomotion by a brace, or by making an arthrodesis at this joint; increasing the power in front of the joint, by transferring the flexor muscles forward or by transferring and uniting the sartorius or tensor vag. femoris to the quadriceps; or in some cases we can act indirectly by stiffening the ankle joint by a brace or by arthrodesis, as in this way the

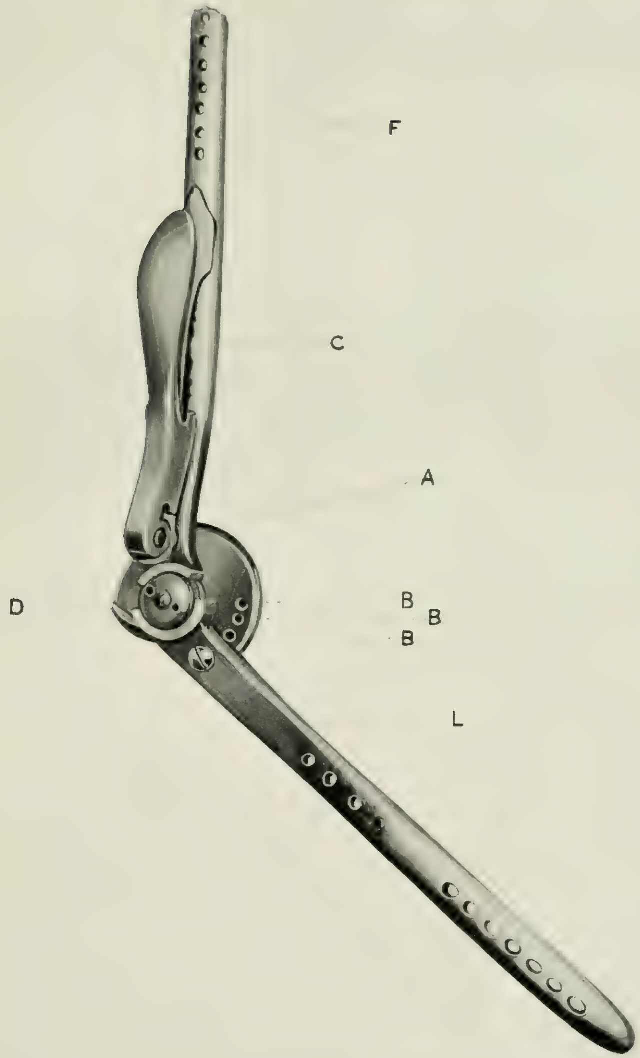

Fig. 2.—A is fixation pin, attached to thigh piece (F) by handle (H), and held in a hole in upper part of disc (D) by a spring (C), unless spring is compressed by patient.

Disc (D) is attached to leg piece by screw (E) in any desired position (B.B.). The joint will be immobilized by spring pin therefore in full extension, or any degree of flexion according to the position in which disc is fixed.

weight of the body in walking is made to pass through a point in front of the axis of motion at the knee, and so tends to extend, rather than flex this joint. If the paralysis has produced a flail joint at the hip, we can stiffen it by a brace, or by arthrodesis, but we must at the same time secure function at the knee, and ankle.

In order that these weakened muscles shall be burdened as little as possible it is necessary that the braces should be made light; they should also be well made, and of the best materials in order to be durable. This makes them expensive, and the cost ranges from ten dollars for an ankle brace to fifty dollars for a brace to support both lower extremities entirely; or we might express it as the cost of from one to five weeks' hospital care.

For this reason much ingenuity has been exercised to devise operations to make braces unnecessary. Hospital care is necessary after these operations for at least six to eight weeks, so that the saving in cost to the hospital is more apparent than real. This argument therefore does not appeal to me so much as the other considerations; relief from the weight of braces, is certainly desirable; the limb operated upon requires some supervision afterwards, but not so much as the braces require to keep them in order; but a stiff joint is stiff forever, while with braces the joint is stiff only when function requires it to be so; this is especially important in the knee and hip joints. It seems to me in considering the best line of treatment to adopt we should always be careful not to do anything which may jeopardize the best ultimate result.

It is remarkable what can be accomplished by means of well adjusted braces. I will give one extreme example in illustration, and may state that I have several patients with just this amount of paralysis who are able to walk about the house, and even get up and down stairs without assistance. Both ankle and both knee joints are without any useful amount of power: one hip joint is in the same condition; at the other hip there is moderate flexion power. The patients are unable to stand, much less walk at all. The brace required for this condition consists of steel bands about the thorax at the level of the scapula angle, and about the pelvis just below the anterior superior spines of the ilium; to these bands are riveted on each side vertical steel uprights, these passing down the outer side of the hip, knee, and ankle joints, and attached to a foot plate, which may be either inside or outside the shoe: for greater strength vertical uprights, attached to the first by thigh and calf bands, also pass down the inner side of the limbs from the middle of the thigh to the foot plate; at the hip joint on the side where there is no power, there is a snap joint, which is rigid in extension when the patient stands, but can be flexed at other times: at the hip which has power of flexion, there is placed a joint allowing flexion and extension, and just below this is a hinge joint allowing abduction and adduction (this last joint allows a little play at this poiut and prevents much wrenching and strain on the brace in the efforts of walking): there are snap joints at both knees, which are rigid in extension when walking but can be flexed at other times: at the ankle joints there are stop joints which allow flexion and extension between 85° and 95° only. When this frame is first applied the child is placed by a bed or table, standing, and encouraged to pull himself by his hands around it; soon he will move the limb with some power to that side, and pull the other after it; then he will balance on this hip and move the other by the trunk muscles; next he will move about with a chair to hold to; later with crutches and finally without any other support than the brace. To go up stairs the stronger foot is swung up the first step, and the other drawn up to the same level by the hands on the balusters: in descending, the reverse movement is made, the rigid limb being lowered first.

Such a brace may be applied after the paralysis has existed five or six months, and as the muscular power gradually returns, limited, or entirely free, motion, may be allowed at the various joints as they are constructed to allow of this. Fig. 1 shows one of these braces: in this case there was no snap joint at the knee. The construction of the snap joint is shown in Fig. 2.

It is a terrible affliction to be compelled to get about in this manner; it is only less than not being able to get about at all. I cannot emphasize this point too strongly. This little power makes the patient a member of society. He need not be constantly attended; he can help himself in innumerable ways and the wonderful change in the mental condition of these patients can only be realized by those who have seen it.

Ophthalmological Division

OPACITIES OF THE CORNEA.—THEIR FREQUENCY IN CHILDHOOD FROM PREVENTABLE CAUSES AND THEIR EFFECT ON VISION.

COLMAN W. CUTLER, M.D.

Ophthalmology has been regarded as a field not distinct from general medicine but comprising problems such as refraction and special surgery which require so thorough a training as to deter the practitioner of medicine. It is obvious that this attitude is deplorable as it tends to make the specialist in reality narrow as well as to deprive the practitioner of information at first hand which is often of great value.

The Ophthalmoscope has become an instrument of precision in the hands of every careful diagnostician. Gower's Medical Ophthalmoscopy, the excellent translations of Haab's Atlases and articles by Bull, DeSchweinitz, Duane, Knapp and by Risley who has drawn attention to the importance of hygiene in the care of the eyes of school children—these writers and many others have awakened an interest in ocular problems. In the care of children, as in no other field of medicine, conditions of nutrition and of the inheritance of a disposition to disease must be anticipated and controlled before the precious moment has passed and the tendency becomes disease.

Some of the special problems met with in aural disease are now recognized by the general practitioner and faced with confidence. The skin also is a field full of suggestion which we study for the most varied signs of general disorder and of lessened resistance to external irritation. We do not need to know the nature of eczema or of the various forms of dermatitis to grasp to some useful degree their significance.

The surface of the eye should be equally patent and if it is studied as part of the surface of the body, containing in its small area some of the same problems met elsewhere, it will yield information equally significant.

In order to make quite clear the nature of the lesions to be considered, it will be necessary to review briefly the structure of the cornea. The surface consists of six or eight layers of stratified epithelium, like the epidermis, and like this, it is renewed if scratched, without the formation of a scar. In reptiles that shed their skin, it is cast off with the external cuticle. Beneath this is Bowman's membrane, the external limiting layer. This is a firm and resistant membrane without apparent structure, which forms a very efficient protection to the stroma beneath from foreign bodies. The resistance of this membrane is evident when a foreign body penetrates the epithelium and lodges in it, and has to be picked out with a needle or when it is necessary to apply some strong antiseptic to an infection of the superficial layers. Without it, the transparency of the cornea would be in constant danger. If Bowman's membrane is destroyed, it is not renewed. Although the epithelium is loosely attached to it, this membrane is firmly connected with the corneal tissue or stroma beneath, which is formed of 50 to 60 layers of fibrous tissue enclosing spaces which contain fixed cells and lymph from the scleral vessels, which forms the only source of nutrition of the cornea.

Posterior to the stroma is Descemet's Membrane, the posterior limiting layer which has upon its surface, in contact with the aqueous humor, a single layer of endothelial cells. The anterior and posterior limiting layers are impervious to fluid and protect the stroma from infiltration from without and from within.

This very elementary sketch may serve to refresh the memory. In dealing with the eyes of children, it is important to remember that, while epithelial damage is not vital, an infection or injury which passes Bowman's membrane will leave an indelible trace, which is a menace to vision if it lies in the area of the pupil.

Before the child is excited by an attempt to separate the lids, the surface of the eye should be inspected if possible by daylight before a window, so that the even surface of the cornea and its reflecting properties may be studied as the eye follows the finger. Any distorting of the bars of the window for instance, will indicate an unevenness, which is a sign of past or present trouble.

The examination of the eyes of a resisting child is not always easy, but if there is a history of eye trouble, or if there is secretion on the lashes, or if the child shuns the light and has evident discomfort and cries when attempt is made to separate the lids, the cornea must be inspected thoroughly. This requires a good light and a lens, not to be used as a magnifying glass, but as a means of focusing the light on the cornea—and any one who has to look at the eye should never be without a lens.

An assistant who can hold the child and steady the head is also needed, although the better plan usually is to hold the child's head in one's lap, steadying it between the knees while the nurse controls the body and arms.

The best elevators are the fingers, but pressure on the cornea must be avoided and this needs practice, especially when the photophobia and blepharospasm is intense, as is often the case in phlyctenular keratitis. If it is necessary to use the elevator of Desmarres, great care must be taken to avoid scraping the cornea.

The control of the child, the arrangement of the light and of the lens, and the manipulation of the lids with due regard for the already inflamed eye may seem difficult, but the importance of these measures cannot be exaggerated. An attempt to describe them in detail may be spared since a very little practice will suggest modes of procedure which will vary with the case. Every book on Ophthalmology deals with this subject. Among many excellent works, Haab's Atlas of the External Diseases of the Eye, edited by DeSchweinitz, will be found especially useful for the general practitioner.

There are three points to be considered in examining the cornea, all familiar but not the less important: the corneal reflex, the smooth surface and transparency. Any clouding of the normal glistening reflex indicates an active process, oedema or infiltration. If, with this, there is a defect or depression of the surface, an ulcer is present in an active stage. If the defect glistens, however, showing that the epithelium is restored, the ulcer is in process of repair, or there is a cicatrix. If the infiltration is deeper, beneath Bowman's membrane, the surface may be intact as in the early stage of a phlyctenule or in interstitial keratitis of syphilis.

The formation of vessels beneath the epithelium in pannus or fascicular keratitis and the deeper vessels in interstitial keratitis are to be noted. Ciliary injection and hypopyon should not escape attention and their significance is well known.

It is the purpose of the writer to impress upon the general practitioner the importance of an early recognition of small lesions of the cornea. In their beginnings, most ulcers can be controlled without serious damage to the eye. Ulcers complicating gonorrheal ophthalmia do not always yield to treatment, however, but here too, an early and vigorous treatment of the disease will usually prevent such unhappy results.

The importance of Crede's method is acknowledged and the immense progress in recent years towards the prevention of blindness is a matter for sincere congratulation. It rests with the practitioner as much as with the ophthalmologist, to see that laws are passed and that incompetent nurses and midwives and ignorant parents are not allowed to lose precious time in temporizing and futile methods before the sovereign remedy, nitrate of silver, is used.

All cases of infants with purulent discharge should be treated as if they had gonorrhea, even if the gonococcus is not found at the first examination. The careful use of nitrate of silver does no harm and may do incalculable good.

The milder silver preparations, argyrol and protargol, should be used as adjuvants only and should not supplant the nitrate of silver in cases that show virulence. In epidemics of measles in a large children's hospital, during the past five years, these milder silver preparations have been used in all cases with secretion, and no such case has developed a corneal ulcer, although previously such complications were not infrequent.

An ulcer of the cornea in gonorrheal ophthalmia may occur at the margin where the swollen conjunctiva overhangs the cornea or near the center where the nutrition is poor. The ulcers at the margin may be due to the accumulation of secretion beneath the swollen conjuntiva which makes it very difficult to inspect and cleanse the cornea without abrading the epithelium. The central infection is due in many instances to injury received during examination or cleansing.

It is not the purpose of this paper to deal with the treatment of these cases, but the importance of a thorough examination, and of care in opening the lids and in removing the secretion cannot be exaggerated.

The nutrition of an infant with ophthalmia is often a problem of greater difficulty than the treatment of the disease itself. In a recent paper on this subject (Lesions of the Cornea in Gonorrheal Ophthalmia and Phlyctenular Keratitis: Am. Jour. Obstetrics. Vol. LIX: No. 4: 1909), attention was called to this important point and reference was made to an article by Lobenstine and Harrar, who demonstrated the ill effects of gonorrhea in the mother on the early nutrition of the child. A question which is often raised is whether the child may remain at the breast. If the infant is nursed by its mother and her intelligence permits proper care being taken to protect her own eyes, this course should be followed, but in an institution, it is rarely possible without assuming a grave responsibility.

Phlyctenular conjunctivitis has been a moot point in ophthalmology as is eczema in dermatology, and the two conditions have been associated as if they were of the same nature. This only adds to the confusion because it brings under one head two conditions of whose etiology little is known and which actually have little in common.

Phlyctenules are small nodules or collections of round cells beginning beneath the epithelium and frequently beneath Bowman's membrane, as has been shown by Baas. They are not vesicles as their archaic name implies. No organisms have been found in the early stages, although staphylococci are numerous in their later development. It is probable that they arise from internal irritation—i. e., endogenously from toxins or, as has been well expressed by Axenfeld, that they indicate the manner in which the conjunctiva of scrofulous persons especially reacts to various forms of irritation.

These eruptions are of importance because they appear insidiously and are often not brought to our attention until permanent damage has been done to vision. They are very frequent, especially in ill nourished children of the poorer classes whose eyes have been neglected, especially in those with adenoids, whose nares are obstructed and crusted, or who have eczema of the scalp and blepharitis. Undoubtedly ulcers of the cornea arise from direct infection from these sources, but the early stages of phlyctenules are quite characteristic and should be regarded as a probable indication of tuberculosis. After a phlyctenule comes to the surface as an ulcer and is secondarily infected, it loses much of its distinctive appearance, but the deeper infiltration and the vascularity are always suggestive of its origin.

In twenty cases examined recently, in addition to several with manifest tuberculosis of bones or glands, a positive reaction to tuberculin was noted in all except two cases, which were superficial ulcers and not typically subepithelial. Axenfeld has made similar observations and Verhoeff whose views of the relation of phlyctenules to tuberculosis have been quoted in the article previously referred to, is strongly of this opinion. These true phlyctenules should then be classed as stigmata of tuberculosis, and as they not infrequently occur in apparently healthy children, they offer an important indication for further investigation and general treatment.

A characteristic of phlyctenules is their tendency to recurrence during any depression of vitality and their disappearance with tonic treatment and improved hygiene. With the great progress that has been made in recent years in school inspection and hygiene, this very important and interesting condition has diminished during the school age, but in the early years it is frequent, and the scars left on the cornea are too often a cause of impaired vision and lessened usefulness in later life. One meets with young men and women who wish to become stenographers or teachers or nurses, and whose intelligence justifies the ambition, but whose defective cornea makes the continued eye work a source of unnatural effort and ultimate discouragement. Many cases of unilateral squint are caused by a minute fleck on the surface of the squinting eye. These local results of the fleeting and often ignored eruption in infancy are very important, and I am glad to be able to call your attention to their broader significance as a sign of a dyscrasia against which we are making a united contest.

Syphilis is a potent cause of corneal opacities and the traces which it leaves are as typical for the diagnosis of that disease as Hutchinson's teeth. In the initial stages of interstitial keratitis, the cornea is intensely vascular, but the vessels are deeper than in pannus following trachoma or scrofula. These deep vessels are believed to be permanent, remaining visible after all infiltration has been absorbed, as fine branching twigs, extending from the limbus into the corneal tissue. They are seen when the pupil is dilated, with a+20D. lens in the ophthalmoscope, if the observer approaches close to the patient and throws the light into his eye. This sign of an early syphilis should be available to the general practitioner as it is sometimes present when other symptoms are absent and may throw light on obscure conditions of nutrition.

To summarize this paper in a few words, the writer has tried to show the importance of familiarity with the appearance of the normal cornea and to define the changes which have a general significance. The methods of examination have been indicated and certain precautions in the manipulation of the lids have been emphasized. The method of examination with a lens by means of oblique illumination is easily acquired and should be practised in all cases where the eyes show signs of irritation and as a means of diagnosis in obscure cases where tuberculosis or syphilis is suspected. Especial stress has been laid on the early recognition of ulcers and infiltrations which, if neglected, may permanently impair the sight.

Finally, if I may burden you with the repetition of a familiar idea: the importance of recognizing latent tuberculosis not only for the sake of the individual, but of society, is obvious.

Scrofula has been a useful term but must give way to a more definite expression. It is not needed where tubercular glands or other lesions are present in which the bacilli may be demonstrated and in other cases which react to tuberculin, and which have phlyctenules or other signs of a toxemia without a discoverable focus, it may be convenient to speak of latent tuberculosis or of tubercular toxemia.

It has been said that most of mankind has successfully resisted tubercular infection and the corollary is, if the condition had been recognized soon enough, the number of those who have not successfully resisted it might have been greatly diminished.

The question of the wider use of tuberculin in schools and especially in children's hospitals is so involved with social and economic considerations that it cannot be dealt with in this paper. Nor does it belong to a discussion devoted to ocular matters, but like most medical problems, it overlaps many fields.

Certain members of the race are the hosts of organisms to which they are in a measure adapted or which they tolerate, but which are still dangerous to others. Unquestionably a very great factor in the dissemination of typhoid are the "typhoid carriers;" so in venereal diseases, those in whom the disease is latent are most dangerous, and in tuberculosis especially no effort should be spared to discover the unsuspected cases. We may doubt whether the prophecy of Prof. Lankester in his Romanes lecture of 1905—"that the knowledge of the causes of disease has become so far advanced that it is a matter of practical certainty that, by the unstinted application of known methods of investigation and consequent controlling action, all epidemic disease could be abolished within a period so short as fifty years" will be realized, but the ideal of Eugenics so recently raised, must be held in mind and all possible effort made to realize it.

Pathological Department

PHAGOCYTOSIS OF RED BLOOD-CELLS AFTER TRANSFUSION.*

J. GARDNER HOPKINS, M.D.

A patient suffering from an extreme anemia, resembling the primary pernicious type, was admitted to the hospital on the service of Dr. Theodore C. Janeway. Immediately following transfusion, smears of the peripheral blood showed great numbers of polymorphonuclear neutrophil leucocytes containing red blood cells. The picture was unique and so suggestive as to certain results of transfusion that it seemed worth reporting.

REPORT OF CASE.

Abstract of History.—The patient was a man of 48, who for eight years had lived in Porto Rico. His health was excellent up to about eight months before his admission, from which time he dated his illness. At first he lost appetite, began to be drowsy and easily fatigued, and lost weight. Later he had diarrhoea, passing mucus but no blood. This had recurred at intervals up to the time he was seen. He had had one period of improvement, but for a month previous to admission he had grown steadily and rapidly worse. He had lost over 35 pounds, more than one-quarter his weight.

Examination.—When admitted he was extremely emaciated. His skin was pale, but not lemon yellow. The physical examination was practically negative. The stools were brown and fluid and contained no blood. After long search three actively motile amebas were found, but no eggs of parasites. The urine contained a faint trace of albumin and a few hyaline casts; otherwise it was normal. The pulse was from 80 to 110; temperature 98 to 99.8 F.; respirations 24. The blood contained 1,350,000 red cells, and 4,000 leucocytes to the c. mm. Hemoglobin 23 per cent; (Fleischl-Meischer.) Color index 0.85. Polymorphonuclears, 49 per cent; lymphocytes, 48 per cent; eosinophils, 3 per cent. The red cells varied greatly, some 3 to 4 microns,

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*Reprinted from the Archives of Internal Medicine.

some 10 to 14 microns in diameter; the majority were large, 8 to 9 microns. There were many poikilocytes, and considerable polychromatophilia, and a few normoblasts were found. The diagnosis lay between a chronic amebic dysentery with extreme secondary anemia and primary pernicious anemia of an irregular type.

Course of Disease.—After admission the patient continued to grow worse. In five days his red cells fell to 850,000, his hemoglobin to 18 per cent, the leucocytes to 2,800, with 56 per cent of lymphocytes. On the tenth day his red cell count was 700,000, his hemoglobin 14 per cent. His condition was so serious that it was decided to try transfusion. His brother volunteered as a donor, and his serum and the patient's serum were tested by Dr. Hans Zinsser for isohemolysins and agglutinins. The patient's undiluted serum agglutinated the donor's corpuscles markedly, and after standing twelve hours in the ice-box there was slight hemolysis. Four other normal individuals were tested, none of whose serums agglutinated or hemolyzed the patient's corpuscles. In combination with the patient's undiluted serum (equal parts of corpuscle emulsion and serum), one volunteer's corpuscles were fairly well agglutinated, another's slightly agglutinated, and those from the other two not at all. There was no hemolysis in any of the tubes on standing in the cold. As it has been suggested that serums may agglutinate when diluted, which do not agglutinate in concentration, dilutions of the patient's serum with 0.85 per cent salt solution from 1 in 2 to 1 in 10 were tested, but showed no agglutination with cells from any of the four sources. Unfortunately the two volunteers whose corpuscles showed no agglutination withdrew, and the one whose cells were slightly agglutinated had to be made the donor.

Transfusion.—This was performed by Dr. H. H. M. Lyle. A free flow was established from the donor's radial artery to the median basilic vein of the patient, and allowed to continue for one-half hour. After the operation the patient had incontinence of feces, and was very restless and somewhat irrational. He bled from the transfusion wound and from a needle puncture in his finger, but the bleeding was soon controlled. About six hours after the operation he became comatose, developed a hemiplegia and died three hours later.

AUTOPSY FINDINGS.

Bone-Marrow.—This was obtained from three or four inches of the middle of the shaft of the humerus and femur only. It was fatty in

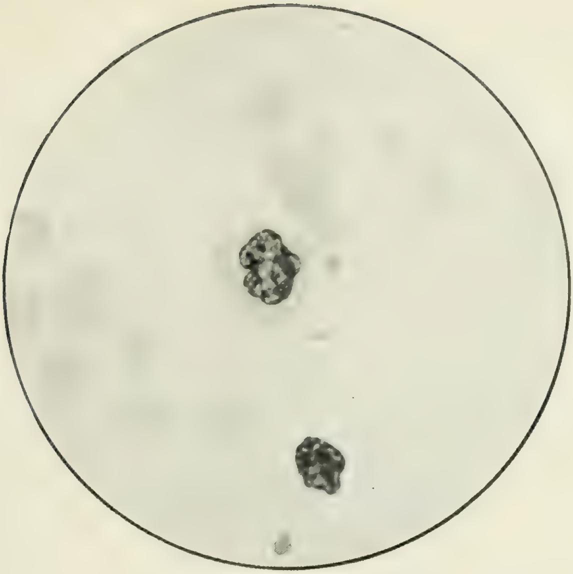

Fig. 1.—Megaloblast from blood after operation.
(Wood stain; × 1,000.)

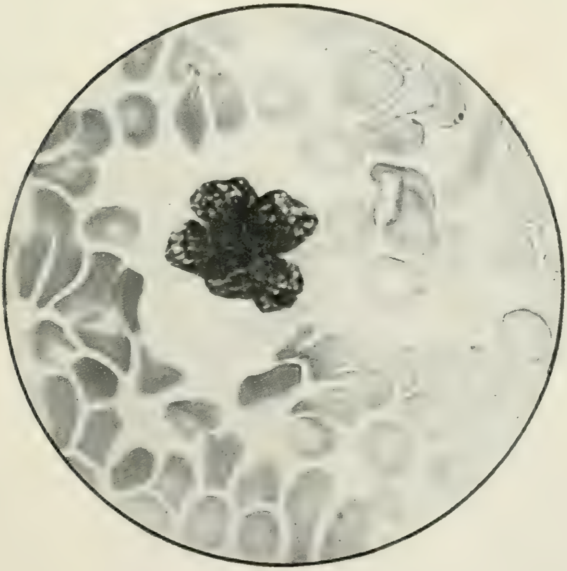

Fig. 2.—Megakaryocyte from blood after operation.
(Wood stain; × 1,000.)

consistence and of a diffuse pink color, with small dark red dots. Sections showed fatty tissue with minute hemorrhages, and small islands of cellular marrow in which myelocytes of various types predominated over the red cells. There were few normoblasts and still fewer megaloblasts. Phagocytic cells containing from two to six red cells were numerous, but these were all of endothelial type with small oval vesicular nucleus. Some polynuclear cells showed yellowish particles, but none were found with distinct red cell inclusions.

Spleen.—This was slightly enlarged, soft and congested. Microscopically, the sinuses were dilated with blood and the pulp packed with red cells. An occasional mononuclear phagocyte containing red cells was found in the sinuses. There were scattered granules of hemosiderin both free and in the endothelial cells and polymorphonuclear leucocytes.

Mesenteric Lymph-Nodes.—These were enlarged. Microscopically, the sinuses were much dilated. In them were numbers of phagocytes containing red cells, most of which were small and pale. Some of these phagocytes were mononuclear; others were polymorphonuclear.

Liver.—This was slightly congested. It showed some fatty degeneration and deposits of hemosiderin, most of which was contained in the endothelial cells. The gastro-intestinal tract was negative except for a few petechiae and some hyperplasia of the solitary lymph follicles. There were no ulcers in the colon and no parasites were found.

BLOOD PICTURE AFTER TRANSFUSION.

The interesting feature of the case was the blood picture after transfusion. The red cells of the recipient rose from 720,000 per c.mm., with 15 per cent hemoglobin, to 1,700,000 with 35 per cent hemoglobin after the operation. The donor's count fell from 5,000,000 to 3,950,000 and his hemoglobin from 95 per cent to 75 per cent Smears of the patient's blood before operation showed no change from previous examinations, but in smears taken immediately afterward, there were found great numbers of phagocytes containing from one to six or more red cells. There was apparently an increase in the number of leucocytes, and a shower of normoblasts and megaloblasts. About 40 per cent were small, normal-looking lymphocytes, but there were also some very large lymphocytes and cells with a large nucleus and an abundant deeply basophilic protoplasm resembling Türck's stimulation forms. Most of the polymorphonuclear leucocytes were normal in appearance, but there were some small forms. There were also a number of very large cells measuring 15 to 22 microns, which stained poorly and resembled the large degenerated forms often found in leucocytosis, except that they were much larger. There were a few pale giant cells, one measuring 22 by 27 microns in diameter, with a nucleus composed of five or six round lobes grouped closely together—evidently a megakaryocyte from the marrow. The phagocytic cells all showed neutrophilic granulation, except those that contained so many red cells that the protoplasm was entirely concealed. Some of these contained a mass of red cells packed so closely together that they could not be counted definitely. The smaller ones had a polymorphous nucleus, which, however, took the Jenner stain very faintly; in others the nucleus was compressed into chinks between red cells. Most of the phagocytes were rather large, from 15 to 20 microns in diameter. The ingested red cells were for the most part round and about 6 microns in diameter. A few were irregular and showed processes projecting into the pseudopods of the captor. They stained evenly, but most of them were pale. All gradations were found, from red cells of normal size to colorless vacuoles of about the same size. Many of the phagocytic leucocytes and others which contained no red cells contained smaller vacuoles.

A differential count of five hundred cells (Jenner stain) gave the following result:

Polymorphonuclear neutrophils	54	per cent.
Lymphocytes	43	per cent.
Large mononuclears and transitionals	1.4	per cent.
Eosinophils	0.2	per cent.
Myelocytes	1	per cent.
Unclassified	0.4	per cent.

Twenty-eight of the polymorphonuclears contained one red cell, and sixteen two or more. In making the count twenty-nine normoblasts and five megaloblasts were seen.

Smears taken after death, which occurred nine hours later, showed no phagocytosis, but normoblasts and megaloblasts were still present in considerable numbers. Many of the leucocytes contained vacuoles which resembled those seen in the postoperative smears.

DIAGNOSIS

The diagnosis was not very certain even post mortem. The case was probably a pernicious anemia in which the complicating diarrhea and rapidly fatal course prevented the development of the typical

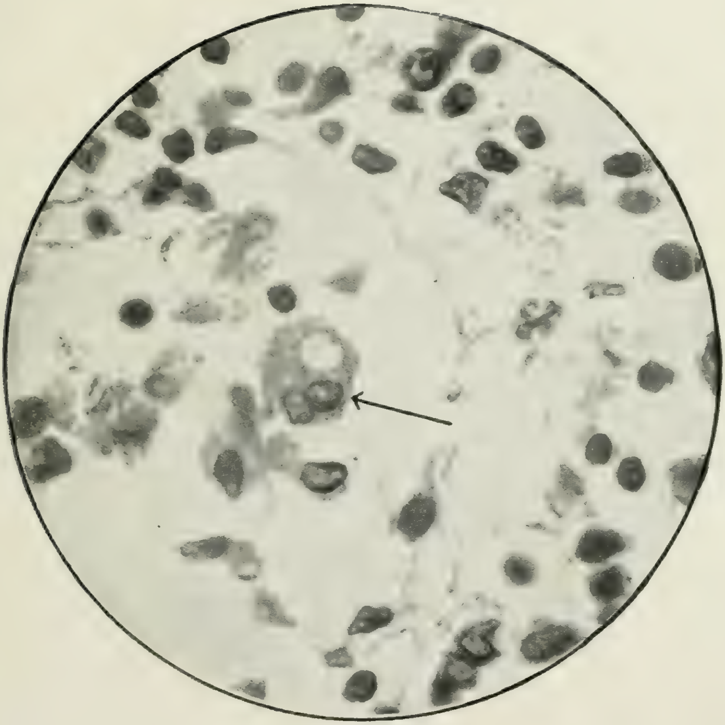

Fig. 3.—Polynuclear phagocyte in sinus of mesenteric lymph-node.
(Hematoxylin-eosin; × 1,000.)

bone-marrow picture. The autopsy excluded any parasitic basis for the anemia and the amebas observed must have been *Entameba coli.* The size of the red cells and final appearance of typical megaloblasts as well as the pigment deposits in the liver and spleen pointed toward pernicious anemia. There must have been megaloblastic marrow in some of the bones to give origin to these cells, and, moreover, even in frank cases of pernicious anemia, the bone marrow may not show hyperplasia throughout its entire extent. The numerous erythroblasts of course exclude the diagnosis of aplastic anemia.

The only other observations of phagocytosis of red cells in the peripheral blood of man, which I have been able to find in the literature, were those reported by Rowley,[1] Bartlett,[2] and Van Nuys,[3] in 1907. They described two remarkable cases of fatal anemia which resembled each other strikingly. In these cases the blood contained numerous large phagocytes, varying from cells resembling lymphocytes to large mononuclear cells, which ingested every variety of normal elements in the blood. In one of these cases the polymorphonuclear cells were also phagocytic and the serum had the power to cause phagocytosis by the large mononuclear cells of normal blood on the warm stage. The white cell counts were very high, 95,000 to 800,000, which would suggest some relation to leukemia. These cases seem to have very little in common with the one just described.

THE MEANING OF THE BLOOD-PICTURE.

I am unable to determine the meaning of the blood-picture in this case. The shape, size, and staining properties of the ingested red cells make it seem probable that many of them were from the donor; while the fact that the phagocytes were so numerous and that many of them were atypical, makes it certain that some at least were from the recipient; so that what was observed was probably a phagocytosis of the transfused red cells by the phagocytes of the recipient. This might be explained in two ways; as a washing out into the peripheral blood of phagocytes from the blood-destroying organs, or as a phagocytosis due to the presence of an hemopsonin in one of the serums.

In regard to the first possibility, there is ample evidence that some such "washing out" of bone marrow elements occurred, in the presence of numerous erythroblasts and the bone-marrow giant cell (which I have found reported only once and that in pernicious anemia[4]). Moreover, there are two factors present which are known to increase the phagocytosis in these organs: pernicious anemia and transfusion.

The increased phagocytosis in pernicious anemia, as is well known, has been emphasized especially by Warthin and by Hunter, who regard it as an essential feature in the pathology of the disease, a process to which the pigment deposits and bone-marrow changes are secondary. Hunter has also found these phagocytes in the portal circulation of animals poisoned with pyrogallol.[5]

As to the effect of transfusion, Gabbi,[6] as far back as 1893, found in transfusing from one normal rabbit to another, an enormous amount of phagocytosis in the spleen of the recipient, which he attributed to plethora. Aside from his work, which was not directed at this problem, there seems to be very little known as to the fate of the donor's cells after transfusion. Boycott and Douglas[7] report that rabbit blood transfused into normal rabbits is destroyed, and that the destruction is more rapid after repeated transfusions. They could not determine the mechanism of this destruction. In the blood charts in Crile's book on transfusion, it will be noted that in the successful cases, the blood-count the day after operation is often higher than that immediately afterward, or in other cases after a fall for two or three days there is then a gradual rise. Weber[8] and others have recently reported excellent results from the injection of small amounts of blood intravenously or even subcutaneously; and it has been suggested that transfused blood does not function directly in the recipient, but acts by stimulating blood formation. Whatever may be the fate of transfused blood in most recipients, it has been noted by Crile and others that in pernicious anemia the count rises temporarily, and then falls rapidly to the previous level or below. The transfused blood is rapidly destroyed in these cases. Phagocytosis in the blood-destroying organs is usually performed by macrophages, but in this case the polynuclears may have taken part in the process and then have been washed into the circulation.

The phagocytosis might also be accounted for by the presence of an opsonin for human red blood cells. Savtchenko[9] found that the injection of inactivated serum of a rabbit immune to guinea-pig corpuscles, together with guinea-pig corpuscles, into the peritoneum of a guinea-pig, was followed not always by hemolysis, but sometimes by phagocytosis. Levaditi[10] found some phagocytes containing red cells in the circulating blood and great numbers in the spleen, after such injections. They attributed this action to the same *substance sensibilisatrice* (*fixateur*) that caused hemolysis, acting in this case in the absence of free cytase (alexin).

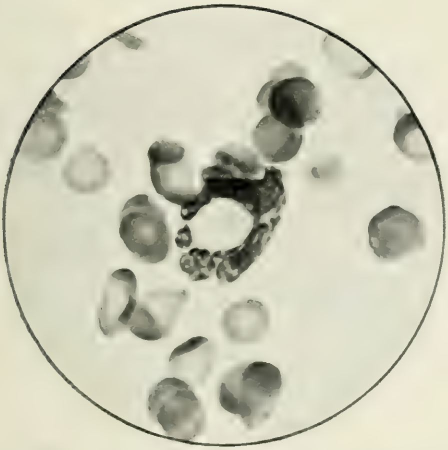

Fig. 4.—Phagocyte with one red cell, showing polymorphous nucleus. (Wood stain; × 1,000)

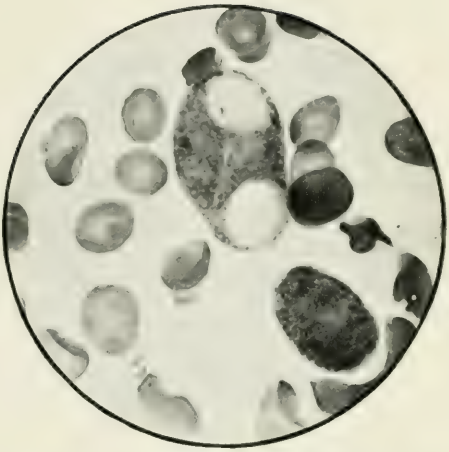

Fig. 5.—Phagocyte with two red cells, showing neutrophil granulation. (Jenner stain; × 1,000.)

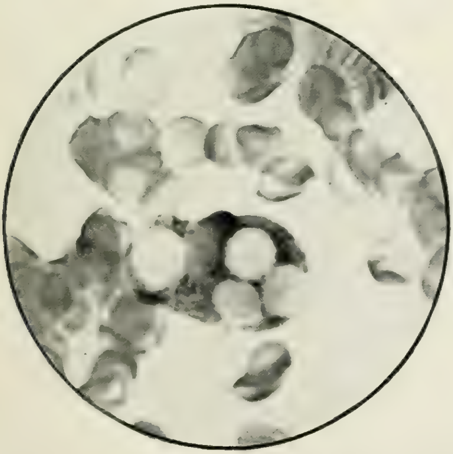

Fig. 6.—Phagocyte with four red cells. (Jenner stain; × 1,000.)

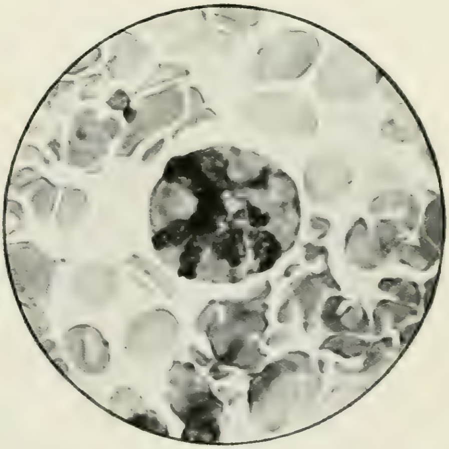

Fig. 7.—Phagocyte distended with red cells, too closely packed to be counted. (Jenner stain; × 1,000.)

More recently, Barratt[11] and Keith[12] studied the same phenomenon *in vitro*, and demonstrated the same substance, which they regarded as a hemopsonin, in inactivated or diluted hemolytic serum. They found this body more sensitive to heat than the hemolytic amboceptor, and also found that it was quantitatively independent of the hemolysins or agglutinins and concluded that it was a distinct substance. Neufeld and Töpfer,[13] working independently, arrived at similar conclusions. Hektoen[14] has investigated the presence of isohemopsonins in human serums. He found them in a number of conditions, especially typhoid fever and scarlet fever; but not constantly in any condition; and it seems unlikely that they have any relation to disease. They occurred independently of agglutinins or hemolysins. Davis[15] and Macgregor[16] found these bodies in cases of cerebrospinal meningitis. Wright[17] reported phagocytosis in two cases of pneumococcus infection, and Eason[18] and later Kämmerer and Meyer[19] have reported autohemopsonins in cases of paroxysmal hemoglobinuria.

The observations of Dudgeon, Panton, and Ross[20] on guinea-pigs injected with hemolytic and cytotoxic rabbit sera show a condition which may have a bearing on our case. In the spleen of these animals they found "an extreme amount of phagocytosis, the large endothelial cells being packed with red blood cells and golden brown pigment." Great numbers of similar phagocytes were found in the mesenteric lymph nodes, a few in the liver and bone marrow, but none in the peripheral blood. This was, of course, an instance of autophagocytosis due possibly to an opsonin in the hemolytic serum.

I obtained serum and cells from this patient post-mortem, but could not demonstrate any opsonin in his serum for cells of several normal individuals, including the donor. The post-mortem serum did not agglutinate the donor's corpuscles. I also tested the serum of two other cases of pernicious anemia, which were negative. The serum of the donor contained no opsonin for cells of a normal individual or for those from a case of pernicious anemia. Serum from a normal individual contained no opsonins for the patient's red cells. These negative results, however, do not exclude the presence of an opsonin in the recipient's serum before transfusion. Such a body would doubtless have been bound by cells of the donor, and it is not surprising that it could not be found free in the serum a few hours later. The agglutinins present when the serum was first examined were evidently bound in this way. The fact that the phagocytes in this case were polynuclears would make it probable that the process was due to an isohemopsonin. In view of this experience it would seem advisable to test for opsonins as well as for lysins and agglutinins in cases in which transfusion is proposed.

In conclusion, I thank Drs. Janeway and Lyle for permission to report this case and for the clinical notes. I also thank Dr. Wood and Dr. Zinsser for their advice and suggestions.

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19 Kämmerer and Meyer, Folia Hematol., 1909, vii, 91.

20 Dudgeon, Panton, and Ross, Proc. Roy. Soc., of Med., 1908-09, Path. Section ii. part 3, p. 64.

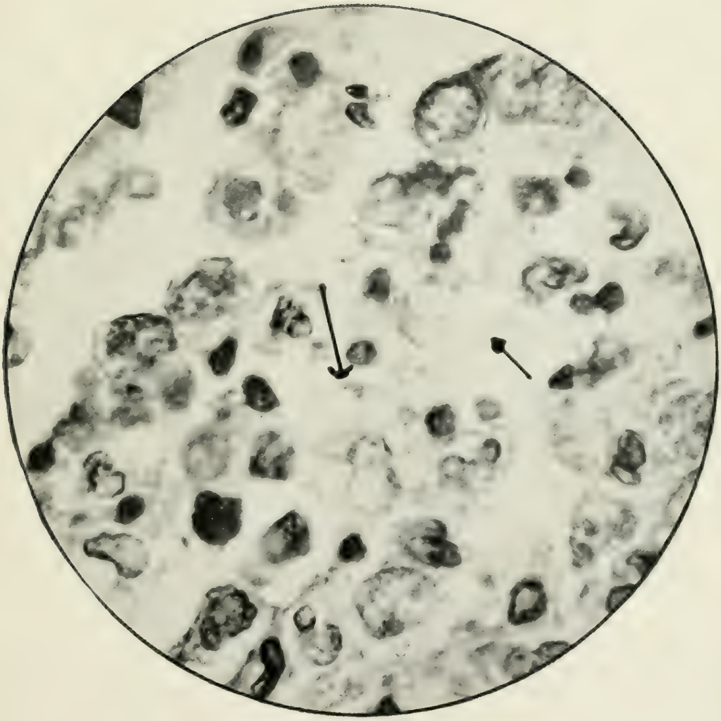

Fig. 8.—Mononuclear phagocyte containing eight red cells, from bone-marrow. (Hematoxylin-eosin; × 1,000.)

ON HEAT-SENSITIVE ANTICOMPLEMENTARY BODIES IN HUMAN BLOOD SERUM.*

HANS ZINNSER, M.D., and W. C. JOHNSON, M.D.

INTRODUCTION.

It has long been well known that normal human and animal blood sera may, under certain conditions, contain elements which exert antihemolytic activity when added to mixtures of red blood cells, hemolytic serum (amboceptor), and complement. The work of most of the writers who have studied these bodies has revealed that normal sera, showing no evidence of antihemolytic action originally, may develop this property when subjected to heat ranging from 50° to 56° C. Such has been the experience of P. Th. Müller (1), Ehrlich and Sachs (3), Noguchi (8), and others. Once formed, moreover, this class of antihemolysins has been found, by some workers, to possess no inconsiderable degree of thermostability, Noguchi finding that 90° C. was necessary to destroy completely the inhibitory properties of such sera.

There is still another class of antihemolytic substances, however, which may appear in normal sera that have not been subjected to heat. Müller mentions the occurrence of such bodies in rabbit and guinea-pig sera studied by him, and attributes their inhibiting properties to direct anticomplementary action. Noguchi, who has studied them in greater detail in connection with his recent work on the "Serum Diagnosis of Syphilis," states that such bodies are not present in fresh human serum, but develop gradually as the serum is preserved, and may be eliminated by heating to a temperature of 54° C. for thirty minutes.

There appear to be two distinct antihemolytic bodies, therefore, which may occur in normal sera. One of these is found in sera after heating, and is relatively thermostable. The other develops in sera on standing, and is destroyed by temperatures which are ordinarily employed for serum inactivation, 54° to 56° C.

The development of specific antibacteriolytic and antihemolytic substances in immune sera previously treated with bacteria or red blood cells, respectively, as described by Pfeiffer and Friedberger (7), and later by Sachs, may, we believe, be ignored, since Gay (5) has satisfactorily shown that the phenomena observed by Sachs did not occur when the corpuscles originally added to the serum had been sufficiently washed. Gay showed that the antihemolytic action observed by Sachs was due to complement absorption by means of specific precipitates formed between the immune serum and the alien serum carried into the mixtures with the insufficiently washed corpuscles.

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The thermostable antihemolytic bodies referred to above have been extensively studied. That their inhibitory action depends upon a directly anticomplementary mechanism, all workers agree. As to the nature of the substances themselves, however, there is much discrepancy of opinion. Müller believed that the antilytic elements were probably present in the native serum but were temporarily masked by hemolytic elements simultaneously present. When the action of the latter had been eliminated by heating, the action of the former became evident.

Ehrlich and Sachs suggest that the application of heat serves, in some cases at least (dog serum heated to 51° C.), to transform the complement into a complementoid with a higher avidity for sensitized corpuscles than that possessed by the complement itself.

Sachs, who attributed the anticomplementary action to complement deviation by free amboceptors of many varieties, assumed to be present in the serum, has apparently been conclusively answered by Gay. His view, moreover, is opposed to recent experience, which seems to indicate that complement is not bound by amboceptor uncombined with its specific antigen.

Noguchi, whose studies of the thermostable antihemolysins of heated sera have been extensive, has come to the conclusion that these bodies are set free in sera as the latter are subjected to heat, more and more being liberated as the temperature is raised above 50° C. At 90° C. he believes that these bodies are either inactivated by conversion into more stable compounds or are masked by the formation of new bodies, "auxilysins," which aid hemolysis. He finds that the inhibitory substances are anticomplementary, active against alien as well as against homologous complement, and that they may be removed from the serum by digestion with red blood corpuscles, the corpuscles becoming thereby more resistant to hemolytic influence. Finally, he states that the antihemolysin, which he names "protectin," may be obtained from such sera as lipoids, or together with these, by ether extraction.

The antihemolysins occurring in unheated sera have been less extensively studied, and it was these bodies particularly to which the attention of the writers was directed.

Occurrence of the Thermolabile Antihemolytic Substances.—The development of antihemolytic substances in unheated human blood sera was noticed by the writers, as it had been by Noguchi, in the course of work with complement-fixation tests. It was observed, also, that the inhibitory bodies did not occur in fresh, active sera, but developed as the sera were preserved at room or refrigerator temperatures, and that, as stated by Noguchi, heating to 56° C. for fifteen to thirty minutes served to eliminate the antilytic powers. After a number of such sera had been studied, it was deemed desirable to ascertain the speed of development of these bodies in normal human serum (animal sera were not investigated), and a series of sixteen normal specimens were examined for their inhibitory powers at intervals of three to five days.

In these experiments .2 cubic centimeter of the serum to be examined was added to .1 cubic centimeter of fresh guinea-pig complement, the volume was brought up to 3 centimeters with salt solution, and the mixture was allowed to stand in a water bath at 40° C. for thirty minutes. At the end of this time 1 cubic centimeter of a 5 per cent. emulsion of red blood cells sensitized with two units of amboceptor were added, the total volume being brought to 5 cubic centimeters. The usual controls were made.

By such experiments it was found that some sera, kept at refrigerator temperature (about 10° C.), would become distinctly antilytic as early as the fifth day, and many completely inhibited hemolysis at the end of two weeks. In the case of two sera not in this particular series, complete inhibitory power, for the quantities given, appeared as early as the fifth day. Other sera exhibited no trace of antihemolytic power after four and five weeks. These sera were examined at the same time for complement, and in every case where no anti-hemolytic substances had developed, free complement could be detected. It is probable that these thermolabile bodies develop in most, if not in all, human sera on standing, appearing as the complement disappears, and that they are originally either not present or masked in their action as long as traces of complement remain.

Two of the sera in which the antihemolytic power developed most rapidly and strongly were taken from patients suffering from chronic nephritis with symptoms of uremia. As this seemed significant, in view of the paper of Neisser and Döring (2), who have claimed the presence of antihemolytic substances in uremic sera, a number of tests were made with sera from similar cases and from two cases of eclampsia. No evidence was obtained, however, which justified any conclusion regarding a definite relationship between the clinical condition and these substances.

In an experiment to determine the temperature at which the inhibitory action was eliminated, sera (after dilution)[1] were heated to 90° C. It was found that most of the antihemolytic action (roughly speaking, about four-fifths) had disappeared. The fact that a trace still remained would tend to show either that the thermolabile and thermostable antilytic bodies may be present at the same time in the same serum or that the latter may be developed in a serum that contained the former.

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[1] In all our experiments the volumes of the mixtures were brought to five cubic centimeters with salt solution to avoid errors resulting from the inhibitory influence of high concentrations of serum, which, as shown by Bordet and Gay (10), oppose hemolysis.

Quantitative Titration of the Thermolabile Antihemolytic Body in Some Sera.—The strength of the antihemolytic power developed in some of the human sera, may be seen in the following titration, in which the serum employed was about three weeks old:

Complement 0.1 c.c. Corpuscles (5 per cent. emulsion) 1.0 c.c. 2 units amboceptor. +	0.2 c.c. serum “W”	No hemolysis.
	0.1 c.c. serum “W”	No hemolysis.
	0.05 c.c. serum “W”	Very slight hemolysis.
	0.04 c.c. serum “W”	Slight hemolysis.
	0.03 c.c. serum “W”	
	0.02 c.c. serum “W”	
	0.01 c.c. serum “W”	Considerable hemolysis, but incomplete.
	0.005 c.c. serum “W”	
	0.0025 c.c. serum “W”	Hemolysis complete.
	0.00125 c.c. serum “W”	Hemolysis complete.

It will be seen, therefore, that quantities as small as 0.005 cubic centimeter of this serum exerted considerable inhibiting action.

Serum “W,” employed in the above experiment, had the strongest antihemolytic action of any in our possession. As early as the fifth day, 0.1 cubic centimeter of it sufficed to prevent completely the hemolysis of one cubic centimeter of sensitized corpuscles.

Does the Antihemolytic Action Bear Any Relationship to Substances Derived from the Red Corpuscles?—Since a number of the sera which showed antilytic properties were observed to be tinged with blood pigment, owing to accidents during separation, one of the thoughts first suggested was that the antilytic bodies might be derivatives of red blood cells, given up to the serum during its separation from the clot. This seemed the more likely because of the observations of Landsteiner and von Eisler (4), Muir (9), and others, that certain red cell extractives did actually exert antilytic action. A considerable number of experiments were made by the writers with this point in view. For the sake of brevity, however, these experiments will be omitted. It is sufficient to say that they showed that both the shadows and products of hemolyzed red cells, when added to a hemolytic system, lent slight antilytic action to sera otherwise not antilytic. These substances exerted, also, a slightly inhibitory action upon hemolysis when added without the presence of human serum. In these respects the experiments confirmed the observations of other writers. In no case, however, did this antilytic action amount to more than a definite slowing of the hemolytic reaction, or, at most, to an extremely slight permanent inhibition. It was impossible, therefore, to attribute the very marked and often complete inhibitory action of some sera, often perfectly clear, to accidental extractions from the clot. For this reason, this course of experimentation was not carried further and is not enlarged upon. It was of use only in again calling attention to the necessity for caution when doing complement fixation tests with sera containing any considerable amount of blood pigment.

MECHANISM OF THE ACTION OF THE THERMOLABILE ANTIHEMOLYTIC BODY.

In speaking of the thermolabile antihemolytic substance, Noguchi stated that its action was anticomplementary. As it seemed to us desirable to verify this, a number of experiments were made.

In one of these a mixture consisting of two units of anti-sheep amboceptor (1–660 cubic centimeter serum in 1 cubic centimeter salt solution), 0.2 cubic centimeter of inhibiting serum "W," and 3 cubic centimeters salt solution, was incubated for one hour at 40° C., and then after the addition of 1 cubic centimeter of a 5 per cent. emulsion of sheep corpuscles, the whole was incubated for one hour at 40° C., and separated by centrifugalization into (1) sediment of red blood cells (subsequently washed three times with salt solution), and (2) supernatant fluid.

To the washed red cell sediment were added 0.1 cubic centimeter of complement and 5 cubic centimeters salt solution. On exposing the mixture to 40° C. for half an hour, the hemolysis was complete. The hemolysis was also complete in the controls. To 3 cubic centimeters of the supernatant fluid (see (2) above) were added 0.1 cubic centimeter complement and 2 cubic centimeters sensitized corpuscles (1 cubic centimeter corpuscles to which two units of amboceptor had been added). On exposing this mixture to 40° C. no hemolysis occurred.

The above and similar experiments showed that the exposure of amboceptor to the inhibiting substance did not prevent amboceptor from becoming attached to the red cells subsequently added. They indicated, furthermore, that exposure of the "sensitized" cells to this antilytic substance did not deprive these cells of the power of subsequently absorbing complement. Neither the different receptors of the amboceptor, nor the complex of amboceptor and red cell were, therefore, in any way altered by the antilytic body, this remaining active in the supernatant fluid. The possibility of explaining the antilytic action as due to the presence of complementoid, was also disposed of by these experiments.

It remained to show that there was no reaction between the antilytic body and the unsensitized blood corpuscle rendering the latter resistant to the subsequent influence of amboceptor and complement.

To determine this point, 0.2 cubic centimeter of antilytic serum "W," 1 cubic centimeter of washed sheep corpuscles (5 per cent. emulsion in salt solution), and 2 cubic centimeters salt solution were mixed, left for one hour at 40° C., and then centrifugalized. The sediment containing the red cells was washed four times in salt solution and then to it were added one unit of amboceptor, 0.1 cubic centimeter of complement, and 4 cubic centimeters of salt solution. On incubating at 40° C. complete hemolysis took place in half an hour (controls hemolyzed in the same length of time). This showed that the corpuscles had not been rendered less sensitive to lysis by exposure to the inhibiting serum. The supernatant fluid from the centrifugalization, containing the 0.2 cubic centimeter of the antilytic serum, was then tested and found to have lost none of its inhibiting power.

This seemed conclusive in excluding the direct action of the antilytic substance upon the corpuscles. Nevertheless, as Noguchi had found that the thermostable antilytic bodies could be absorbed by blood corpuscles, if allowed to digest with them at 20° C. for about eighteen hours, it seemed desirable to determine whether the thermolabile body studied by us would be similarly absorbed. To test this question, the following experiments were made on two separate occasions, each time in triplicate.

One cubic centimeter of the antilytic serum was mixed with 4 cubic centimeters of a 5 per cent. suspension of washed corpuscles, and this mixture was left at 18° to 20° C., in one case for twenty, in another for twenty-four hours. At the end of this time the fluid and corpuscles were separated by centrifugalization. The corpuscles were then thoroughly washed, and they and the supernatant fluid were tested as in the preceding experiment.

The result showed (1) that the corpuscles had not absorbed the antilytic principle, for they had not become more resistant to lysis, and (2) that the supernatant liquid had retained its antilytic property undiminished.

As we have thus eliminated the action of the antilytic body upon the corpuscles, the amboceptor, or the corpuscle-amboceptor complex, its action upon the complement remained as the only possibility. This has been proved in a considerable number of experiments which consisted in exposing complement to the antilytic serum for varying periods, and then adding sensitized corpuscles. Complete inhibition of hemolysis has been the uniform result of such a procedure. That the anticomplementary action of the body in question is almost instantaneous, or, at least, more rapid than the attachment of complement to the antigen-amboceptor complex, was shown by the fact that complete inhibition resulted when antilytic serum, complement, and sensitized corpuscles were simultaneously poured into a test tube and immediately incubated.

It was also ascertained that this anticomplementary action could be overcome by gradually increasing the relative amount of complement in the mixtures. Thus, 0.2 cubic centimeter of the antilytic serum

"W" completely inhibited hemolysis in the presence of 0.2, 0.4 and 0.6 cubic centimeter of fresh guinea-pig complement, when the cells employed were sensitized with two units of amboceptor. Eight-tenths cubic centimeter of complement, however, caused considerable hemolysis in such mixtures, and one cubic centimeter produced almost complete lysis.

The foregoing experiments thus show, in perfect agreement with the statements of Noguchi, that the mechanism of the action of the thermolabile antihemolytic body is an anticomplementary one.

It seemed of interest to make sure that this anticomplementary action was direct, and not indirect, in the sense that it might depend, (*a*) upon the absorption of complement by one or a number of antigen-amboceptor complexes preëxisting in the serum, or (*b*) upon complement absorption by a precipitin reaction occurring between the antilytic serum and either the guinea-pig serum of the complement or the rabbit serum of the amboceptor.

The first supposition, namely, that the complement is deviated or absorbed by antigen-amboceptor complexes preëxistent in the serum, is difficult to approach experimentally. It seems unlikely, however, that this supposition is correct, for in the experiments cited above, in which the antilytic body, sensitized corpuscles, and complement, in excess of the minimum quantity for complete lysis, were mixed simultaneously, absolutely no hemolysis took place. It does not seem reasonable to believe that in such a case *all* the complement should immediately be taken up by the preëxistent antigen-antibody complex, leaving none available for the hemolytic system. Furthermore, it must be remembered that the sera containing the antilytic principle were not inactivated and, when fresh, contained some excess complement. If antigen-antibody combinations had been present, they would, in these cases, be already supplied with complement, and their reaction complexes would be complete. Moreover, the thermolability of the inhibitory substances (which become inactive on heating to 56° C.), is also against this view.

The second suggestion, namely, that complement was deviated by precipitin reactions taking place between the antilytic serum, on the one hand, and rabbit serum or guinea-pig serum, on the other, was disproved by the following experiments:

(*a*) A precipitin reaction between the inhibiting serum and the rabbit serum of the amboceptor was excluded by sensitizing corpuscles and subsequently washing them very thoroughly (four times) in salt solution. The subsequent addition of antilytic serum and complement resulted in complete inhibition, showing that after the removal of all traces of rabbit serum no diminution of the anticomplementary phenomenon had taken place.

(*b*) The possibility of a precipitin reaction between the guinea-pig serum of the complement and the antilytic serum was eliminated by the use of complement from other sources. Thus, in the following mixtures:

1.	Inhibiting serum	0.2 c.c.	Left at 40° C. for 15 minutes.
	Human complement	0.2 c.c.	
	Salt solution	3.0 c.c.	
2.	Inhibiting serum	0.2 c.c.	Left at 40° C. for 15 minutes.
	Rabbit complement	0.2 c.c.	
	Salt solution	3.0 c.c.	
3.	Inhibiting serum	0.2 c.c.	Left at 40° C. for 15 minutes.
	Guinea-pig complement	0.2 c.c.	
	Salt solution	3.0 c.c.	

The subsequent addition of sensitized corpuscles resulted in complete inhibition, while control tubes with similar varieties and quantities of complement showed hemolysis. There was one exception to this, which we did not follow in detail. One antilytic serum did not seem to oppose the action of dog complement. We mention this, in passing, without trying to explain it.

It seems reasonable, from the foregoing facts, then, to conclude that the inhibiting action of our serum was due to direct anticomplementary action, using the term merely as differentiating this action from the indirect modes of eliminating complement considered above.

CHEMICAL NATURE OF THE THERMOLABILE ANTICOMPLEMENTARY BODY.

Our first attempt toward the identification of this anticomplementary body with any of the serum components, was based upon Noguchi's success in identifying the thermostable anticomplementary body with the serum lipoids. In our tests, serum diluted ten times with salt solution was shaken for fifteen minutes with ether. As the ether did not readily separate from such a mixture, centrifugalization was then employed, and the supernatant ether was carefully pipetted away. Reëxtraction with ether in the same way was twice repeated. The serum residue was then examined for inhibiting powers, the quantities employed being similar to those which inhibited completely in the original serum. Such experiments showed that absolutely no loss of anticomplementary action had been sustained by ether extraction. It seemed unlikely, therefore, that the serum lipoids, at least those extractable with ether by this rather crude method, could account for the anticomplementary action of our serum.

In order to ascertain whether any of the dialyzable components of the serum were responsible for the anticomplementary action, ten cubic centimeters of diluted anticomplementary serum were dialyzed through parchment for forty-eight hours against frequently changed distilled water. Again, the serum residue, which became turbid, probably by the precipitation of globulin in this process, showed absolutely no loss of its antilytic power.

It remained to examine the serum proteids. Five cubic centimeters of the serum were diluted with forty-five cubic centimeters of salt solution, and an equal bulk (fifty cubic centimeters) of a saturated ammonium sulphate solution was added. The precipitated globulins were removed by repeated filtration through a triple thickness of fine filter paper. The filtrate was dialyzed against running water to remove the ammonium sulphate. The substances on the filter paper were scraped off, redissolved in an 0.85 per cent. solution of sodium chloride, and reprecipitated with ammonium sulphate. Finally, they were dried and rapidly rinsed with cold water to remove gross particles of ammonium sulphate. Allowing for losses during this process, the bulk of the globulin precipitate was taken up in thirty cubic centimeters of an 0.85 per cent. salt solution. This solution was tested in varying quantities roughly representing from 0.1 to 0.2 cubic centimeter of the original serum. Complete inhibition of hemolysis resulted. The filtrate after forty-eight hours of dialysis still contained traces of ammonium sulphate, but possessed absolutely no anticomplementary action. These tests were controlled with ammonium sulphate solutions in order to preclude the possibility of hemolysis due to the ammonium sulphate alone, and with ammonium sulphate solutions plus the original antilytic serum, to preclude the possibility of interference with the inhibiting principle on the part of the ammonium sulphate.

It appeared from these experiments that the antilytic principle had been removed from the serum with the globulins.

It now seemed desirable to ascertain whether the antilytic factor of the globulins obtained from the serum was thermolabile, as was the antilytic principle in the original serum.

To determine this point, 1 cubic centimeter of the globulin solution (representing approximately 0.1 cubic centimeter of the original serum) was placed in each of four test tubes, and in each of four others was placed 1 cubic centimeter of the original antilytic serum, after this had been diluted ten times with salt solution. Each of the tubes containing globulin was paired with one containing the antilytic serum, and the pairs were subjected to heat for twenty minutes, one to 50° C., another to 56° C., another to 60° C., and another to 70° C. To each tube was then added 0.1 cubic centimeter of fresh guinea-pig complement and 2 cubic centimeters of salt solution. The mixtures were allowed to stand at 40° C. for thirty minutes and sensitized corpuscles were then added.

The result showed that there was a complete parallelism between the globulin solution and the original antilytic serum, neither being affected by 50° C., but the antilytic action of both being removed by temperatures between 56° C. and 60° C. It thus appeared that the thermolabile substance could be removed from the antilytic serum with the globulins, and that the globulins, precipitated from such serum and redissolved, displayed anticomplementary and heat-sensitive properties exactly like those of the original serum.

CONCLUSIONS.

The work recorded above has served to corroborate the observations of other writers, notably of Noguchi, that in many human sera, if not in all, there develop, on standing, anticomplementary bodies which are delicately thermolabile, being inactivated by heating to 56° C. for fifteen to twenty minutes. It has appeared that these bodies may occasionally be present in sera which, after heating, may develop the thermostable anticomplementary body referred to by other authors. It has become evident also that the speed of development of the thermolabile anticomplementary body in different sera is subject to much variation. The thermolabile body appears as the complement disappears. The question arises whether the thermolabile anticomplementary body may not be originally present in the serum, temporarily masked by the native complement. This would seem improbable from the fact that titration has shown that 0.1 and 0.2 cubic centimeter of the inhibiting serum will often inhibit as much as 0.6 cubic centimeter of fresh guinea-pig complement, a quantity superior to the amount of complement present originally in the antihemolytic serum. It seems, therefore, that the anticomplementary body must actually be formed in the serum during the period of preservation.

The thermolabile antihemolytic body studied by us is like the thermostable body investigated by Noguchi, in that it inhibits alien as well as homologous complement; it is unlike the thermostable body, however, in that it cannot be absorbed from serum by digestion with red cells, nor does it render the treated cells more resistant to hemolysis.

The thermolabile body may be removed from inhibiting sera by precipitating the globulins. A solution of the globulins then manifests a thermolabile anticomplementary action.

No relationship between a clinical condition and the appearance of these bodies in the sera, has been found.

As a practical result these studies have shown, as have those of Noguchi, that the complement fixation tests should never be made with certain sera which have been preserved for some time without inactivation.

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[2] A thorough review of the literature of the subject may be found in Noguchi's article, The Thermostable Anticomplementary Constituents of the Blood, *Jour. Exper. Med.*, 1906, viii, 726.

LABORATORY DIAGNOSIS—ITS IMPORTANCE TO THE PRACTITIONER.*

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As we near the end of the first decade of the new century, there is everywhere noticeable a most remarkable tendency in medicine to criticize, sift and otherwise take stock of our present knowledge; even concerning the value of our system of medical education there is much searching of hearts. There is a general feeling that we are approaching a new era in medicine, and an expectation that the twentieth century will see further great discoveries which will rival the magnificent achievements in surgery of our profession during the nineteenth century. Such an aura is not always followed by the expected results, but if the rate of progress of the past ten years is continued, the hope is certainly justifiable.

While the medicine of today is, theoretically at least, extraordinarily in advance of that of fifty years ago, yet there are men now in active practice in this state, the beginning of whose training dates back to that period, and we all, no doubt, gladly acknowledge that they are physicians of ability, that they diagnose and treat their patients' troubles successfully, and do as much as, if not in many cases more than, the graduate of the past ten years. It is evident for this and many other reasons which need not be enumerated here, that much of our accumulated scientific knowledge of disease must be regarded as merely a preliminary basis for the future work of investigators in medicine, and that this century is to see the expansion, synthesis, and practical application of the scientific facts discovered, or about to be discovered, to the direct healing of the sick. For this reason there is now a strong call for the laboratory investigator to turn from abstract problems and to concern himself with the immediate needs in hand: the cure of the sick. It certainly is in answer to this call that the great Rockefeller Institute and its hospital for medical research have been established in New York; that George Crocker gave his millions for the study of cancer; that the Phipps Institute for the study of tuberculosis is growing and extending its influence in Philadelphia; that money has been given to study the hookworm question and to check the ravages of the parasite; that the United States government through its admirable army and navy medical services is investigating the diseases of the tropics in the Philippines and in Panama. It is because of their response to this call that Reed, Carroll and Lazear are honored in the history of American medicine, their lives sacrificed for the direct benefit of the human race.

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It has occurred to me that the members of this section may be interested in some of the conclusions forced upon me by contact with this movement in medicine, a movement which threatens to put every physician into a laboratory and to make every laboratory man if possible a practicing physician; to fuse the methods of clinical observation with those of the microscope and test tube; to regard each phase as equally important in some cases, each as valueless in others. It is even beginning to place the treatment of a few of our patients in the hands of skilled laboratory men who alone are capable of handling some of the most potent and also the most dangerous of our therapeutic agents.

The so-called laboratory methods can be divided into two classes; one, those which every practitioner can and should employ; the other, those which by their very complication or their demands on skill, time and apparatus, necessitate the employment of a special worker with elaborate training. If we run over a few of the simpler tests which the practitioner himself may use, you will notice that they are not all the most recent. In fact, a good rule for such methods is that which some one gave for books: Never read a book until it is at least a year old. For laboratory tests I think the time should be extended to at least five years. During that time the matter will be sifted and looked at from all sides, and a large series of observations will be collected, so that we can really and finally determine the practical value of any method. We have been deceived so often in the past that one is inclined to be skeptical when a new thing appears. Generally the mode of selection is by simply dropping a test. One hears less and less of some novelty, until finally it disappears from the standard textbooks and is heard of no more.

This change in attitude is very evident in the use we make of the examination of blood. When I entered the hospital as an interne, leucocyte counts were in vogue, and many weary hours we spent in looking through a microscope, enumerating the white corpuscles, no matter what the disease from which the patient was suffering. Today any medical student can tell you just how much and how little value the leucocyte count has. He knows, for example that there is a group of rare diseases—the leukemias—in which the diagnosis can be accurately made only by the examination of the blood and the determination of the number and variety of the leucocytes. In such cases the man trained in laboratory methods must make the diagnosis. We all know also that in infection a large number of leucocytes indicates in general a good resistance, while a low count shows the reverse. But we also know that in pneumonia some patients recover with a very low count, while others die with a high one. We are aware that a high count often indicates pus; but we know that a low number of leucocytes by no means excludes an extensive abscess. We have in the past ten years discovered that the relative count of the white cells is in many cases more important than their total increase; that a high polynuclear percentage suggests an acute infection or hemorrhage, and that one over 95 per cent is rarely seen except in pneumonia. But we also know that patients without an acute infection may occasionally show a high polynuclear count. In other words, the surgeon should not be called upon to open an abdomen solely on the number and variety of the leucocytes. Useful as these laboratory determinations may be, the patient's face, his pulse, his temperature, his physical condition, his history, must all be taken into consideration in making the diagnosis. The leucocytes are only one phase of the whole question.

Let me pause for a moment to cite a case in point. A young man was brought into the hospital ward suffering from a high continuous fever, with great prostration. He had a slight bronchial cough, with no sputum. As he said that he had been sick only a few days, he was supposed to have typhoid fever, and was put on typhoid diet; and the laboratory was requested to make a routine blood examination, aWidal test, and a blood culture. The leucocytes were high, about 12,000, with a relatively high polynuclear count. The Widal test was negative as was the blood culture. Inasmuch as in the early stages of typhoid fever, organisms can be isolated from the blood in about 98 per cent of the cases by the use of proper methods, and as at that period of the disease a low leucocyte count and relatively high lymphocyte count are the rule, the laboratory sent back word that the case was not one of typhoid fever. The attending physician remarked to his staff that he was born long before the laboratory was heard of. The laboratory, somewhat irritated, I must confess, thereupon took up the study of the case, and found that the patient's ward history was not full; that he had been coughing and losing weight for two months. After considerable difficulty some sputum was obtained, which on examination showed numbers of tubercle bacilli. The man at no time had typhoid fever, and he is now doing well under fresh air treatment for tuberculosis. Physical signs appeared at the right apex at about the same time that the bacilli were found in the sputum.

It seems to me that there is considerable material for meditation in a case of this type, which shows that one must be ready to appreciate the relative importance of the phenomena which we are studying and to remember the weight which should be given to each clinical symptom. In typhoid fever the blood culture and the Widal have a far higher value than mere prostration and fever, as the following figures from Treupel will show, though these were compiled in 1905, a time when technique of the isolation of bacteria from the blood was not as perfect as it is now. The proportionate diagnostic value of the symptoms in percentages, is:

		Per cent.
1	Isolation of bacteria from blood	93
2	Widal reaction	91
3	Slow pulse in proportion to fever	87
4	Clinical history permitting diagnosis	75
5	Diazo reaction in urine	74
6	Rose spots	72
7	Palpable spleen	57

In pneumonia, on the other hand, the most important thing in diagnosis is to hear evidences of consolidation in the lung.

To return to our subject: we know that certain changes in the red cells are indicative of a fatal anemia, the so-called pernicious type. We also know that a similar picture can be produced by a species of tapeworm, fortunately rare in this country. We also know that a blood condition very closely imitating pernicious anemia can be induced by repeated hemorrhages, especially from hemorrhoids and ulcers of the intestine. We know that certain types of anemia are accompanied by leucocytosis which aids in determining the nature of the disease. We know that other types are not, which again points a way for treatment.

The demonstration of the organisms of malaria in the blood is of more importance than any other symptom of the disease. In all acute untreated cases the parasite can be demonstrated by a sufficiently careful search.

We have learned to examine urine with more intelligence than formerly; to separate temporary albuminurias and cylindrurias from nephritis. We know now that the diagnosis of eclampsia and Bright's disease is not made by the determination of urea in the urine. As a matter of fact, most of such determinations are not worth the paper they are written on. The freezing point and conductivity tests are no longer in general use. We know that the estimation of uric acid in the urine does not make a diagnosis of gout. Strange to say we are returning to the old thread test for uric acid in the blood, which Garrod recommended thirty years ago. Glycosuria no longer of necessity means diabetes, nor acidosis approaching coma; and a yellow skin and conjunctiva are as important as a bile reaction in the urine.

We have learned that the stomach does not secrete a mathematically constant amount of hydrochloric acid or of ferments, also how much the emotions influence that secretion, and how useless are elaborate tests without taking nervous instability and the general conditions of the body into full account. A good deal of palpation and some inflation of the stomach may be worth much more than chemistry in individual cases. We have learned to simplify our tests and make them more accurate. We know how extremely important traces of blood in the feces are for the diagnosis of ulceration or new growth along the gastrointestinal tract. We have learned of how little value most of the other chemical tests on the feces are. The eggs of parasitic worms and the amebae of dysentery are the chief diagnostic findings from the side of morphology.

In the sputum, if we can demonstrate tubercle bacilli and differentiate them from other organisms, we have almost covered the ground of practical diagnosis; gross inspection at the bedside will do the rest. The general practitioner is beginning to analyze milk and to study the effect of modifications of that important fluid in regulating the diet of children under his care without calling in the pediatrist.

Of bacteriological methods there are but two which the practitioner need constantly employ. These are the microscopical examination for the diphtheria bacillus, the tubercle bacillus, and the meningo-and gonococcus, and the agglutination test for typhoid fever. In the large cities, fortunately, the municipal laboratories will make any or all of these tests free of charge; but it is often important in private work or when one is at a distance from facilities to be able to make at least a good preparation and examine for these common organisms. It frequently enables the practitioner to save a patient from blindness from gonorrheal ophthalmia and enables him to decide upon the nature of a vaginal discharge in little girls, which we have gradually learned is most frequently gonorrheal. It may save twenty-four hours in the isolation and treatment of a case of diphtheria when time is most precious. The demonstration of the tubercle bacillus in the sputum may definitely save a life by permitting the necessary hygienic methods to be installed before the disease has involved a large portion of the lung.

The Widal reaction for agglutinins in the blood of persons suffering from typhoid fever is one of the borderland tests which it is difficult for the average practitioner to carry out unless he has a fairly good laboratory training, and I think it will ultimately be done entirely in central laboratories by specialists. The technical value of the Widal is at present settled. It is one of the most valuable symptoms in typhoid fever, appearing more often in the course of the first ten days than any other individual symptom, if we except bacteremia and fever. On the other hand, it does not tell us that the disease is typhoid much before the end of the first or the beginning of the second week, and if the case is well marked a clinical diagnosis should be obtained before the Widal becomes sufficiently strong to enable a positive report to be given. In the obscure, irregular cases, however, of which we see so many, it is our most valuable diagnostic symptom, with the exception of blood cultures. If we leave out these two tests we still employ in the diagnosis of typhoid fever the methods of our fathers and grandfathers.

Blood cultures belong to the second group, which the practitioner himself can not employ as a usual thing. Except in typhoid fever they do not as a rule afford a sufficiently early diagnosis to permit of beneficial therapeutic attack; and excluding this disease hardly 10 per cent of cases of acute endocarditis or sepsis give positive results. When a patient has streptococci or staphylococci circulating in the blood, that patient is as a rule doomed, and as yet none of our remedies seem to be of much value. The colloidal metals wax and wane in popularity. Polyvalent streptococcus sera apparently work wonders in one case and do nothing in the next. The vaccines in generalized infections seem to do harm rather than good. Indeed, if there were any scientific basis for their application they would be contraindicated in a patient whose entire system is flooded with organisms. In judging the value of such remedies it is in these very cases that extreme caution must be exercised. I have never forgotten the lesson which we all received in New York some years ago from the report of a surgeon who claimed to have cured a case of streptococcus septicemia by the injection of formaldehyde into the veins. The patient did indeed have streptococci in the blood and did improve under the formaldehyde injections; but apparently this was only an accident, for of the many persons who received formaldehyde treatment afterward, but few survived. It was promptly shown by experiments that the injection of formaldehyde into the veins of animals suffering from bacteremia hastened their death rather than otherwise. A single case, therefore, is absolutely of no value in judging of any therapeutic agent —a fact which can not be too frequently reiterated.

Concerning the newer reactions with sera or bacterial products we are still in the course of accumulating data to enable us to judge of their practical value. These tests form a transition between those which are purely practical and may be applied by the physician, and those which belong more especially to the laboratory, though the attitude is gaining ground that they should by no means be generally employed by any one who has not had thorough training in the methods of bacteriology. The old subcutaneous method of applying tuberculin carries with it possible danger and too much discomfort to the patient to be generally available, though its value in determining obscure tuberculous lesions in nonfebrile patients is unquestioned.

I also venture to prophesy that the Calmette conjunctival reaction will soon fall into disuse, as there are already more than fifty cases now on record and many others not recorded in which vision has been seriously damaged by the drug. If a physician tells his patient that one person in ten thousand loses his sight from the use of the Calmette test, the patient is apt to retort that he will wait and let the doctor make the diagnosis in some other way rather than submit to the danger. The same information can be obtained by the skin reactions without the attendant risk.

The von Pirquet tuberculin test suffers from the defect that a large proportion of adults react to it, and while we all know that almost every grown person has or has had a small tuberculous lesion, it does not help us in the diagnosis of the present case to have a reaction due solely to old lesions. What we want to know is whether a catarrhal bronchitis is tuberculous, or whether it is of a simple nature. We want to know whether there is an active process going on and not that there is an old partially healed lesion at the apex of one lung. The von Pirquet reaction, therefore, in its present state of development is of little value in diagnosis, except in the study of young children. Here it promises to be of great value especially as pointing the way to hygienic procedures to prevent the access of the tubercle bacillus to the lungs, while the child is unable to protect itself from the sputum of its parents or the milk of tuberculous cows. Von Pirquet's paper showing the great rise in percentage of tuberculous disease, less than 1 per cent being due to bovine bacilli, from infancy upwards, is instructive as indicating how early tubercle bacilli enter the body, though his statistics are not true for America because of the extraordinary prevalence of tuberculosis in the Viennese population, with which he worked.

The Moro salve by which tuberculin is rubbed into the skin is less apt to give a positive response to small quiescent lesions than pure tuberculin, and seems to be, as far as present observations go, of much more value than the Pirquet. The same is true of Detre's method of inoculation with tuberculin, though it is doubtful whether the interpretation which he places upon slight differences in the type of reaction as he employs it will stand criticism. Certainly the Moro and Detre reactions are not as a rule given unless some active tuberculosis is present, though they are, as is well known, often absent in very active cases, such as those of miliary tuberculosis.

We will now turn to an entirely different group of tests, those in which the practitioner obtains only the results, the methods being too complicated and time-consuming to permit one who is in practice to carry them out. This group contains a number of complicated and difficult procedures based upon the researches in immunity carried out by Metchinkoff, Bordet, Wright, and Wassermann, and many others. The opsonic index of Wright as a means of diagnosis is, it seems to me, rapidly losing ground in New York and elsewhere. In treatment also we are rather inclined to neglect the index as unreliable, and as extremely difficult and time consuming to determine. Careful observations of the patient's general condition are usually quite sufficient to enable us to carry on treatment with tuberculin or the so-called vaccines or suspensions of dead bacteria. In New York there is a quite evident tendency to place the use of tuberculin therapeutically in the hands of a few special workers who have had a large experience in the employment of the drug, and the general practitioner is rather giving up the use of this most dangerous remedy.

The vaccine treatment, too, is being placed in the hands of specialists, chiefly men who have had long bacteriological training, and who understand how to make their initial cultures, how to sterilize the massive growths, and how to dilute them previous to inoculation, and what is equally important, the type of infection which ought to be benefited. I think that the indiscriminate use of so-called stock vaccines in the therapeutics of acute infections cannot be too strongly deprecated. We are injecting into the blood current particles which may form emboli or thrombi, and may give rise to the most serious constitutional disturbances by the toxins which they carry in their bodies or give off to the surrounding culture media. A number of examples of serious damage done to patients by the inoculation of large quantities of vaccine in unsuitable cases are now on record.

We can not be too cautious in judging from a few cases the value of some new methods of treatment. A friend of mine has related to me how he thought that he had obtained a new vaccine for pneumonia, and how the first thirty patients treated by the mixture survived, many of them running a short course apparently aborted by the inoculation. Thirty successfully treated cases of pneumonia in a mixed hospital material in New York is an extraordinary occurrence. But after the thirtieth case there came a similar run of fatal cases, in which the inoculation seemed to produce not the slightest effect, and the final outcome was that little or no benefit was obtained from the vaccine. The same thing was noted after the application of leucocyte extract by Hiss and Zinsser, where the results in pneumonia, while they at first seemed astonishingly good, afterward indicated that the extract was of little use in the very cases which needed help; in other words, it was valueless in severe cases, and of use only in those cases which were likely to get well without much assistance.

The diagnosis of rabies in the animal and the treatment of that disease in the human being has assumed considerable importance of late, owing to the ease with which the Negri bodies can be demonstrated in the brain substance of all animals or persons dying from this disease, and the therapeutics of the disease have been put upon a new basis. The demonstration of these organisms, however, is a matter of considerable difficulty and requires special training. The treatment also should be left in the hands of those members of the state and local boards of health who have had to do with the preparation of material for inoculation.

Another laboratory test which is becoming more important to the physician, much more so to the surgeon, is the test for the hemolytic activities of the blood of persons who are to undergo transfusion. I can not insist too strongly upon the necessity for preliminary examination of the blood of the person who is to act as donor, to see whether hemolysis is produced, and I do not share the generally expressed opinion of the surgeon that as long as the patient is moribund, transfusion may as well be done, running the risk of a fatal hemolytic reaction. The physician often has to decide whether a patient is to be transfused or not, for they are not all surgical cases, and it is especially important that the type of anemia for which the transfusion is to be done should be carefully determined. An experience of my own has taught me to regard not only the hemolytic bodies, but also the agglutinative substances. In a recent transfusion in which very slight hemolysis and agglutination was observed, the patient died in a few hours from cerebral thrombosis due probably to the formation of agglutinative thrombi, while in the circulating blood were found large numbers of phagocytes evidently destroying the corpuscles which had been injected into the patient's circulation. The reason for this phenomenon was probably the peculiar qualities of the blood seen in type of anemia from which the patient was suffering. It should by this time be well known that the infusion of blood in true megaloblastic pernicious anemias rather hastens the inevitably fatal end, while in the secondary anemias beneficial results may be expected.

I am not sure, however, that transfusion will be regarded as an important procedure in the future, for everything now points to the fact that the red corpuscles given by the healthy donor are to a certain extent foreign bodies, or at least useless particles to the recipient. The chief thing he gains from the corpuscles is a stimulation of his bone marrow. If that blood-producing organ is beyond any stimulus, no result is obtained. The patient, in fact, has extra labor in getting rid of the residue of the corpuscles. On the other hand, the injection of fresh serum from the rabbit or horse seems to work extraordinarily well in certain types of purpura and progresssive hemorrhage. You all know that in many of these cases the coagulation time of the blood is absolutely within normal limits, and that calcium lactate and other drugs have no effect. The injection of 10 to 20 c.c. of rabbit serum under the skin will often check such a hemorrhage, especially in the new-born. I have been interested in watching the babies at the Sloane Maternity Hospital improve under this simple procedure. The same method has been of benefit in postpartum hemorrhage, and I doubt if the dangerous and difficult operation of direct transfusion will long survive.

When, in 1905, that brilliant young zoölogist Fritz Schaudinn saw under the microscope the Spirochete pallida in the fresh serum from a chancre, the study of syphilis entered upon a new era, for the diagnosis of the disease has been greatly aided by the fairly easy demonstration of this parasite in primary and early secondary lesions under suitable conditions. We will not argue here whether the Sp. pallida is the causative agent in syphilis or not. We know that it is constantly found in that disease in man and animals, it may be in symbiosis with some invisible organism; but for the present the finding of this little corkscrew parasite in the serum from an ulcer is sufficient evidence to warrant beginning treatment.

I said fairly easy, because I think the demonstration of Sp. pallida is exceedingly difficult, merely because of the minuteness of the parasite. It is now perfectly possible to stain it in the course of a few minutes by any one of half a dozen stains. One of the simplest and best of these methods is that which I published some years ago as a stain for the malarial parasite.[1] Thin smears of the serum squeezed from the lesion to be examined are fixed for three minutes in methyl alcohol, and flooded with a 1 to 1—1000 aqueous solution of eosin w. g. (Grübler), using about 0.5 c.c. of the eosin stain on the smear. A 1 to 400 aqueous solution of methylene azure blue I (Grübler) is dropped directly on the slide and mixed with the eosin, using about the same quantity as of the other dye. The proportions seem to be relatively unimportant. It is essential only to get an excess of the blue. A few trials will show how many drops should be used. This mixture is left on the slide for six to eight minutes and is then washed off with a strong stream of tap water; the slide is dried and examined with an oil immersion. The Sp. pallida is stained a deep red, as are the nuclei of the leucocytes. The bacteria and other spirochetes usually assume a bluish tone, which is a great advantage in distinguishing between Sp. dentium and Sp. pallida.

The parasite may also be found with the microscope arranged for dark-field illumination. This is not very easy unless one is trained in the examination of syphilitic material, for what is apparently Sp. pallida can be frequently demonstrated in scrapings from the teeth of perfectly normal individuals. This Sp. dentium, which occurs in all our mouths, resembles very closely the specific spirochete, except that it is shorter and has fewer and shallower turns to the spiral. Of course, in the serum from a secondary lesion of the skin or from a chancre, no such confusion is apt to occur, but in secondary lesions from the throat great care must be exercised in making a diagnosis.

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[1] Medical News, lxxxiii, 248.

The easiest method of all is the ink method of Burri. The spirochetes can be easily demonstrated in the exudate by mixing with it a little India ink of fine quality (Bourgeois or Gunther Wagner is the best), and allowing the preparation to dry in a thin film. The spirochetes are sharply outlined and can usually be identified without difficulty, if they have a sufficient number of turns to distinguish them from the Sp. dentium. Very short specimens of pallida, however, can not be sharply separated from dentium by this procedure.

At almost the same time as the discovery of the organism came the announcement that Wassermann had discovered complement fixation in the serum of syphilitics. This is perhaps the most important of all the laboratory tests which have been devised in the past ten years. Originally worked out by Bordet and Gengou as a purely biological phenomenon, Wassermann and his pupils applied it to the diagnosis of syphilis. They were pleased beyond measure to find that apparently the reaction was specific. Shortly after, when a general study of the subject had been begun in the various laboratories of Europe, it was pointed out that fatty or lipoidal substances were those which played the part of the syphilitic immune body. Subsequently it was shown that the blood giving this reaction had no immunizing power in animals. In other words, it did not contain an immune body, but a chemical substance produced in the tissues and found in excessive quantities in the circulating blood of persons suffering from syphilis. Thus, an experiment which apparently had ample scientific foundation and which was successful, was afterwards found to be based upon entirely erroneous conclusions, and, therefore, the only method of determining its final value was to collect a large series of observations showing the incidence of the positive reaction in syphilitics and its absence in healthy people.

It is now four years since Wassermann, Neisser and Bruck carried out their first experiments on syphilitic apes and obtained this so-called reaction of complement fixation. This is not the place to go into the details of this test. It is the most difficult, most complicated, and most puzzling of laboratory procedures, and no one can do it accurately without long training. The recently published monographs of Bruck in Germany and of Noguchi in America render any detailed description here unnecessary. Some twenty thousand cases are now recorded which show that positive results are practically never obtained in normal human beings, and only in syphilitic or parasyphilitic conditions. For example, in Bruck's study of 5028 control tests only fifty-nine were positive, and if certain doubtful cases are thrown out, the rate falls to less than one in a thousand. While the reaction may occur occasionally in leprosy, or very rarely in carcinoma, and possibly in scarlet fever and certain protozoal infections, yet as the technique has improved the number of these conflicting reactions has decreased, and many of them are undoubtedly due to imperfect technique.

We now know that in the early stages of the disease, we get a moderate number of positive reactions, depending of course upon the extension of the disease. We know that practically all cases with secondary lesions give a positive reaction if they have not been treated. We know that many of the late cases give reactions and that the ones which do not are the ones which have undergone thorough treatment. We know that practically all cases of general paresis give the reaction, and that about 50 per cent of cases of tabes give it. Evidently, tabes is not so absolutely dependent upon syphilis as has been thought.

The chief difficulty in interpreting the results is the fact that treatment inhibits the reaction, and we have not as yet sufficiently extensive statistics to enable us to say just how much treatment may check the reaction, or what time must elapse between the treatment and the reappearance of a positive test. This question must be cleared up in the near future in order to permit us to obtain the greatest benefits from the test. A case will illustrate: I examined a middle aged man who gave a negative reaction. He had a hard obscure thickening of the cord, and epididymitis, with a large hydrocele. On tapping the hydrocele there still remained a firm tumor. The surgeon who had sent him to me thought that the negative reaction must mean carcinoma. On investigating the patient's history, I found that he had had a small amount of treatment a few months before. This had consisted merely of a few tablets of mercurous and potassium iodide. A vigorous course of antisyphilitic treatment did not affect the local condition and an operation was considered advisable. At the time of operation I made a microscopic examination of a section. The hydrocele sac was immensely thickened and inflamed and from a fragment I was able to make a positive diagnosis of syphilis. The surgeon felt that considering the patient's age, the fact that he did not respond to treatment, and the possibility of a new growth, he had better remove the affected testicle. On incision of the testis it was found to be entirely occupied by a gummatous mass. The absence of a reaction after slight treatment, therefore, does not necessarily prove the absence of a syphilitic taint.

The following case illustrates another phase: A small specimen, which had been removed from the pharynx of a patient in one of our large hospitals, was sent to me. I reported it as epithelioma. The clinician retorted that the case was evidently syphilis from a clinical point of view, and that I must be mistaken in my diagnosis. I asked him whether thorough antisyphilitic treatment had been applied, and he said that it had and volunteered the information that the Wassermann reaction was also negative. This, however, was made light of because, the clinician said, the effect of treatment was to nullify the test, and that it really was therefore of no value at all. I retired as gracefully as possible, and afterwards saw the patient in a moribund condition from extension of the new growth from the pharynx into the lymph nodes of the neck, showing that the growth had been an epithelioma all the time.

It is also very dangerous to tell a patient, on the basis of a blood examination, that he has not syphilis. People are constantly applying to the laboratory, having heard of the new test, and are asking to have it done. They often will not state whether they have had treatment or not. Under such circumstances a negative reaction is not of the slightest value; in fact, it is often misleading. On the other hand, a negative reaction in an untreated case may save people from much worry and it is especially of advantage in quieting some of the syphilophobes who fill our cities owing to the indiscriminate spread of imperfect knowledge through the current press and by some of our well meaning physicians. I recently had a man and his wife apply to me for a Wassermann. Neither had any suspicion of syphilis, but some physician had made a diagnosis of syphilis on the wife. She knew enough to accuse her husband, and the situation was unpleasant. As neither had had any treatment a negative reaction from both was a great comfort to the family.

The immense value of the public health and life insurance side of the question is evident. In Dresden, for example, in the Nursling's Hospital it was found that 10 per cent of the wet nurses gave a positive reaction, and that three-fourths of the children who had been nursed by these women gave evidences of syphilis. The time will probably come, therefore, when no wet nurse will be allowed to fulfil her most important function without a clean bill of health as far as the Wassermann reaction goes. The regulation of women of the street will also be much easier when we have a positive means of determining whether they are syphilitic or not. I do not doubt that life insurance companies will soon demand a Wassermann test, at least for their selected low rate cases. This has already been suggested in Germany, and when we think of how important a factor syphilis is in the causation of diseases, such as aneurism, aortic regurgitation, arteriosclerosis, and cerebral hemorrhages, we can see that the life insurance companies would do well to make special rates for those who give a positive reaction. Of course, here, the knowledge that treatment will inhibit the reaction, which will soon be widespread, will prevent full use of the method.

It has been necessary to give merely a superficial sketch of the many technical methods which the practitioner may employ. A full discussion of any one of these would have occupied my hour. My reason for this form of presentation was a feeling that the members of the society would gain more benefit from hearing one whose work in medicine has been largely confined to the laboratory procedures express the plain truth in regard to these methods and point out that they do not solve more than a few of the questions which face the physician when he is called upon to make a diagnosis. We must never forget that the most of medicine is still in the trained eye and the skillful fingers, in the eliciting of an accurate history and the balancing of the facts so obtained. I do not doubt that in 90 per cent of the cases which the physician sees in his practice, the diagnosis may be very well made without the application of any but the simplest tests, such as the hemoglobin blood test, and the albumin and sugar test in the urine. But the practitioner who is not interested in laboratory work should be trained to note such cases as are beyond his diagnostic ability, and if he is to be honest with his patients they must be sent to some one with such training. The practitioner who attempts to treat cases of diabetes without a fairly full knowledge of his patients' metabolic peculiarities, is wronging those patients. There is a large group of cases of disorders of the stomach and intestine which need the information which a test meal can afford despite history and careful physical examination. It is rarely possible to make a diagnosis of carcinoma of the stomach in time to enable the surgeon to be of any benefit to his patient, without some of these special methods. Here lies the advantage of the men who have made a special study of such conditions, and while I fully realize some of the disadvantages of specialism, yet I believe in it most firmly.

The question arises, however, how is a busy practitioner to obtain the benefits of laboratory knowledge. It is manifestly impossible for a man who spends twelve or fourteen hours of his day in seeing patients to keep in training or to read up on special methods. He is too tired when his regular work is over, or he may never have had the opportunity to get the necessary preliminary scientific training. It has seemed to me that the only ways in which the practitioner can obtain accurate and satisfactory reports from laboratory tests is, not to attempt to do them himself, but either to adopt the old preceptor system, using a graduate in medicine instead of an undergraduate, or to arrange with several of his colleagues to support some recent graduate who will devote himself for a few years to running a small laboratory. Under the first scheme the younger man would have the benefit of the physician's long experience and could get a training comparable to that given by the hospitals to post-graduates. This method is being used by many of my confrères in the East, who arrange with promising young men that they shall do all of the laboratory tests on private patients and either share the fees or get a regular salary. The scheme of a central laboratory is perhaps more economical, because one man can then do the laboratory work of six or eight practitioners. If a reasonable guarantee is offered to such a man he can fit himself by taking a course on the more difficult tests, such as the Wassermann. It is perfectly possible for a man to make a good living if sufficient practitioners support him with their laboratory work, and the success of a number of my old students in this field is an evidence of its practicability.

Even if the physician may not be blamed if he is indifferent to laboratory methods from the point of view of diagnosis, he can not with impunity treat those suffering from bacterial diseases without definite knowledge of the infective organism. For this reason all the newer antitoxic methods, such as those employed for the meningococcus, and the vaccine therapy introduced by Wright, require an absolutely accurate identification of the inciting organism, before injection of the patient with these most powerful and dangerous drugs. It is never difficult to convince the patient of the need of these tests in order to get him to pay for the very best information, and I think that in the future the general medical practice of this country will be improved only by a closer union between the laboratory man and the practitioner in one of the two ways which I have outlined. The time has passed when any one individual can cover the entire subject of general medicine.

CLINICAL ANALYTICAL METHODS.

KARL M. VOGEL, M.D.

The following directions for performing the ordinary analyses used in clinical laboratory work are published, not because they contain anything original, but because they are the ones employed in St. Luke's Hospital laboratory and in the classes in clinical pathology in the College of Physicians and Surgeons, and have been in use for a long enough time to show that they are sufficient to instruct a beginner so that he can carry out the methods with reasonable accuracy. They form thus a set of standard analytical methods which may be of interest to a wider circle than the workers in the hospital laboratory.

USE OF THE BALANCE.

See that the balance is perfectly level, as indicated by the plumb bob; if it is not, make it so by turning the adjusting screws at the right and left front corners.

Always place the object to be weighed on the left-hand pan, and the weights on the right-hand pan. One reason for this is to avoid errors through possible inequalities in the length of the two arms of the beam. To obtain the true weight of a substance when extreme accuracy is required, counterbalance it with sand or weights and then replace the object with weights; or weigh in one pan and then exchange the substance and weights, weigh again, and take the mean of the weights so obtained.

The beam and pans must always be supported before adding or removing weights, and the weights must be handled only with forceps.

To avoid errors in noting weights always count them twice: 1st, by noting those missing from the box; 2d, by noting the weights as they are taken from the pan and replaced in the box.

All objects must be at room temperature when weighed. Warm objects can not be weighed accurately as currents of air are caused which introduce an error.

A platinum crucible should remain in the desiccator ten to fifteen minutes, and a porcelain crucible twenty to twenty-five minutes before weighing.

The supports of beam and pans must be lowered gently to avoid injury to the knife edges.

The balance case must never be left open or with the beam unsupported, and the rider must be removed from the beam.

Be careful to avoid spilling the substance to be weighed on the pans or on the floor of the balance case. If this happens, remove at once by dusting carefully with the camel's hair brush.

Liquids must be weighed in closed weighing bottles, and solids in weighing bottles or watch glasses.

From time to time determine the zero point of the scale. Set the pans swinging by producing a current of air against them by moving the hand palm down briskly downward in front of one pan, and then close the door of the case. Designate the central division of the scale as 10, and determine the points on the scale to which the pointer moves on either side after the first two or three swings. Take at least two readings on one side and one on the other, and obtain the mean; e.g.:

Left	Right
5.6	13.6
6.0	
2)11.6	13.6
5.8	13.6

$$\frac{5.8+13.6}{2}=19.4 \qquad \frac{19.4}{2}=9.7=\text{zero point}$$

To determine the tenths of a milligram, either use a rider or, if great accuracy be required, estimate them by interpolation, as follows:

Weigh to the nearest milligram too light, and obtain the zero point as described above. Call this reading Y. Add 1 milligram and again obtain the zero point, calling this Y^1. Call the zero point of the balance unloaded A. Then the fraction of a milligram to be added to the weight of the object will be equal to

$$\frac{Y-A}{Y-Y^1}$$

VOLUMETRIC METHODS.

Burettes: With colorless solutions, read the bottom of the meniscus. With colored solutions, such as permanganate, read the top of the column of fluid. To avoid errors through parallax keep the eye at the level of the top of the column of fluid. Solutions should always be at the standard temperature engraved on the volumetric apparatus when they are measured.

Keep burettes covered with caps when not in use, and always use a funnel to fill the burette, but remove it before adjusting the level of the fluid.

As the absolute error of reading remains constant, the percentage of error is greater the smaller the amount of solution used; and the quantity of liquid employed should, therefore, preferably not be less than 20 to 25 c.c.

Burettes and pipettes must be free from grease or the solution will not moisten the surface of the glass evenly and will collect in droplets. To clean burettes and pipettes, use a mixture of 100 c.c. concentrated sulphuric acid to which 1 gram of potassium bichromate has been added. Fill the burettes with the mixture, allow to stand for some time, and then wash carefully in water. Finish by rinsing with distilled water.

Pipettes are ordinarily made to discharge the amount indicated by the graduation; measuring flasks to contain it. In both instances the bottom of the meniscus is the point read.

The opening of the pipette should be small, so that from ten to twenty seconds will be required to empty the instrument. Allow the point to touch the side of the receiving vessel as the last drops flow out.

The upper end of the pipette and the finger used in closing it must be dry in order to control the flow of liquid properly.

If corrosive fluids are to be measured with a pipette, always slip a piece of rubber tubing 20 to 30 cm. in length over the end of the instrument to avoid the possibility of getting fluid into the mouth. Never draw ammonia into a pipette with the mouth.

INDICATORS.

These are bases, acids, or salts, which change color in the presence of acids and alkalies respectively, and possess so intense a color as to enable small amounts to tinge relatively large quantities of fluid.

The indicator itself takes part in the reaction between the acid and alkali; that is, salts are formed or are decomposed into their respective bases and acid radicles. It is this alteration in chemical constitution that gives rise to the change in color.

The indicators in most common use and their leading characteristics are as follows:

Indicator	Color with Acids	Color with Alkalies	Use with Carbonic Acid in Cold Sol.	Use with Carbonic Acid in Hot Sol.	Use with Ammonium Salts	Use with Organic Acids
Litmus	Red	Blue	Un.*	Re.	Re.	Re.
Methyl orange	Pink	Yellow	Re.†	Un.	Re.	Un.
Phenolphthalein	Colorless	Pink	Un.	Re.	Un.	Re.
Cochineal	Purple-red	Blue	Re.	Re.	Re.	Un.
Alizarin red	Yellow	Red	Un.	Re.	Re.	Re.
Congo red	Blue	Red	Re.	Un.	Re.	Un.

*Unreliable. †Reliable.

STANDARD SOLUTIONS.

As ordinarily employed, the term "normal solution of an acid" is used to indicate a solution of such a concentration as to contain 1 gram of replaceable hydrogen per liter; e.g. normal HCl solution contains the molecular weight of HCl (36.458) per liter; but normal H_2SO_4 contains only one-half the molecular weight, because H_2SO_4 has two replaceable H atoms. Similarly, the concentration of a normal solution of an alkali is such that the amount of the base present corresponds to the amount of acid in the normal acid solution; e.g., normal NaOH contains 40.058 g. of NaOH per liter, this being its molecular weight; but of sodium carbonate only one-half the molecular weight would be taken, as one molecule of this base is capable of replacing two atoms of acid hydrogen.

Since oxygen (atomic weight 16) is bivalent, oxidizing solutions are made on the basis of 8 grams of available oxygen per liter. For example, in potassium permanganate ($KMnO_4$, molecular weight 158.15) five atoms of oxygen of the eight contained in two molecules of the salt are available for oxidizing purposes; i. e. 80 parts by weight; therefore, one-tenth of this double molecule (2 x 158.15) or 31.63 grams must be taken for the normal solution, and 3.163 for the decinormal solution.

PREPARATION OF $N/10\ H_2SO_4$.

Take of Merck's anhydrous reagent sodium carbonate about 7 to 8 g., and ignite gently in a previously weighed platinum crucible, not allowing the heating to exceed a dull red in order to avoid the conversion of small amounts of carbonate into hydroxide, which may take place at high temperatures. The object of the heating is to dehydrate the salt completely and to decompose any bicarbonate which may be present.

Allow to cool in the desiccator, and on the balance quickly remove enough to leave exactly 5.3 g. in the crucible.

Dissolve this in hot distilled water, rinsing the crucible well. Allow the fluid to cool and then make up to exactly one liter.

Take 7 c.c. of chemically pure H_2SO_4 (sp. gr. 1.84), dilute with four or five volumes of distilled water, and allow to cool.

Transfer to a two liter Giles' flask, and add distilled water to the 2200 c.c. mark. Shake well, and fill a 50 c.c. burette with the acid, and another with the sodium carbonate solution, in each case rinsing out the burette first with some of the solution.

Measure 50 c.c. of the carbonate into a beaker, add a few drops of methyl orange, and titrate with the acid until a pink tinge is noticeable and the addition of a drop of alkali restores the neutral color. Repeat until duplicates are obtained differing by not more than 0.1 c.c.

The acid will be found to be too strong and the amount of water to be added is poured into the flask after reducing the quantity to exactly 2 liters. The amount of water is calculated as follows: for example,

49.5 c.c. of acid neutralizes 50 c.c. of the alkali; then

$$C=\frac{N \cdot d}{n} \qquad C=\frac{2{,}000x\ .5}{49.5} \qquad C=20.2$$

C = No. of c.c. of water to be added.

N = Amount of solution remaining.

d = difference between no. of c.c. theoretically required and no. of c.c. actually used.

n = No. of c.c. used in titration.

Repeat the titration and correction until the two solutions are adjusted so as to balance evenly. If the acid is too weak, it is simpler to make it a little too strong again by adding a drop or two of concentrated acid, and then diluting to the required degree.

PREPARATION OF N/10 SODIUM HYDRATE.

Weigh out about 10 g. of pure NaOH*. Dissolve in water, and dilute to 2200 c.c. in a Giles' flask.

Standardize with the N/10 H_2SO_4 in the manner described above. For work in which Congo red is to be used habitually it is advisable to employ this indicator in adjusting the alkali to the acid.

Keep in a rubber stoppered bottle.

KJELDAHL METHOD.

Principle: By digestion with concentrated sulphuric acid in the presence of an oxidizing agent, all nitrogenous constituents in the organs, fluids, or excretions of the body are converted into ammonia and the organic bodies decomposed. Potassium sulphate facilitates the reaction by raising the boiling point of the acid. The ammonia is bound by the acid and forms ammonium sulphate. This is then liberated by the addition of alkali and is distilled into a measured amount of decinormal acid. The amount of acid so neutralized may then be determined by titration with decinormal alkali.

Reagents: Concentrated sulphuric acid.
Potassium sulphate.
$CuSO_4$.
30% NaOH.
N/10 H_2SO_4.
N/10 NaOH.
Congo red.
Pumice.

Technique: Measure 5 c.c. of urine with a graduated pipette into a Kjeldahl flask of 800 c.c. capacity. Add 10 c.c. of H_2SO_4 and a small crystal of copper sulphate, and heat over wire gauze in a fume closet until white fumes are given off. Then add a couple of grams of potassium sulphate and continue the heating until the fluid has lost all yellow color, and then for about one-half hour longer. Allow the flask to cool, and carefully and slowly dilute the contents with distilled water to about 400 c.c. Add about a teaspoonful of powdered pumice, and for each 10 c.c. of sulphuric acid used, add 60 cc.. of 30% sodium hydrate without shaking, and avoiding contact of the alkali with the mouth of the flask, as otherwise it is difficult to make the rubber stopper stick tightly. The flask is immediately connected with the safety bulb of the distilling apparatus. An Erlenmeyer flask of 500 c.c.

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*Merck's reagent sodium hydroxide purified by alcohol is satisfactory.

capacity has previously been prepared with 50 c.c. of decinormal H_2SO_4, 10 drops of Congo red, and a little distilled water, and adjusted under the condenser so that the tip of the glass safety tube of the latter dips below the surface of the fluid. The distillation is started (at first with a small flame) and continued for at least an hour. The drops falling from the condenser are then tested with litmus. If still alkaline, the distillation is continued. Otherwise the receiving flask is lowered, the distillation continued for a few minutes longer, and the flame extinguished. The safety tube is detached and is rinsed with distilled water inside and out into the receiving flask. The fluid is then titrated with N/10 NaOH, the end reaction being a brilliant red. In dealing with untested reagents a blank determination is necessary to determine their possible nitrogen content, and this amount should be deducted from the result.

Each c.c. of N/10 acid neutralized =0.0017 g. of NH_3 and 0.0014 g. of N.

The usual amount of nitrogen on an average diet is 15 to 16 g. in 24 hours.

MELTING POINT DETERMINATION.

Draw a piece of glass tubing into a capillary having a bore of .75 to 1. mm.; cut off a portion of this 8 cm. in length; and seal one end. Introduce a small quantity of the substance to be tested by pressing the open end of the tube against the powder and then tapping it down or pushing it in with a fine wire. Attach it to the thermometer with a small rubber band, in such a way that the powder is at the level of the bulb of the thermometer, and the rubber band is as high up as possible. Immerse the bulb of the thermometer in concentrated sulphuric acid in a test tube, which is then heated in sulphuric acid in a beaker of Jena glass or, preferably, in the special Thiele apparatus, and heat with a small Bunsen flame. Watch the substance and note the temperature at which it suddenly becomes transparent or contracts into a droplet. The heating should be slow. If a beaker is used, stirring is necessary to equalize the temperature. At least three determinations should be made.

To correct the temperature for the error due to the exposed portion of the mercury thread, use the following formula:

$S = T + n\ (T - t) \cdot 0.000159.$

S = corrected melting point.

T = observed melting point.

n = length of exposed portion of thread in degrees.

t = temperature of air surrounding the exposed air as determined by another thermometer. Ordinarily the room temperature may be used.

SPECIFIC GRAVITY—WESTPHAL BALANCE.

Clean the glass float and platinum wire, by immersing for a moment in concentrated sulphuric acid. Rinse with water, and dry carefully with filter paper.

Fill the cylinder with urine, and bring this to the required temperature of 15° by immersing the cylinder in water of a suitable temperture. Adjust the balance with the leveling screw at the foot so that it is in equilibrium with the float hanging in air.

Immerse the float in the cylinder of urine, adjusting the height of the balance so that the float is completely covered, but only a little of the platinum wire dips below the surface of the fluid when the beam is horizontal. Hang the first rider on the same hook as the float, and then bring the balance into equilibrium by hanging other riders on the various notches. The riders on the beam give the specific gravity to four decimals, the integers following in the order of the size of the riders and being indicated by the numbers of the respective notches.

PYCNOMETER.

Weigh the pycnometer empty, and then fill with distilled water at 15°, drying the outside carefully with filter paper, and weigh again. Weigh again filled with urine at 15°. Deduct the weight of the instrument in each case, and obtain the specific gravity by dividing the weight of the urine by that of the water.

TOTAL ACIDITY.

Principle: Determination of the acidity of the urine by direct titration is not possible owing to the occurrence of calcium and ammonium salts in the presence of monobasic phosphates. The addition of potassium oxalate, however, overcomes these difficulties by holding in solution the di- and tricalcium phosphates and preventing dissociation of the ammonium compounds.

Technique: To 25 c.c. of urine add 15 to 20 g. of powdered potassium oxalate and several drops of phenolphthalein.

Shake for several minutes, and titrate at once with N/10 sodium hydrate until a distinct permanent pink is obtained, shaking the flask during the titration.

To neutralize a 24-hour amount of urine from 550 to 650 c.c. of N/10 alkali are usually required.

FREEING URINE FROM ALBUMIN AND THE GRAVIMETRIC DETERMINATION OF ALBUMIN.

Take 100 c.c. of urine. If necessary make it faintly acid with dilute acetic acid, and heat on the water bath until the albumin begins to separate in flakes. After drying the outside of the beaker boil for two minutes over a free flame.

If the albumin does not coagulate well, carefully add a drop or two of dilute acetic acid. Excess of acid may cause some of the albumin to stay in solution.

Filter while still hot through a Schleicher and Schüll ash-free filter (No. 589, 9 cm.) that has previously been dried at 120° and weighed. If further quantitative determinations are to be made on the urine, filter into a measuring flask and wash out the beaker and filter with small amounts of distilled water until the volume at room temperature is brought up to its original amount. Wash precipitate on the filter with more warm water and then with alcohol followed by ether.

Dry in the hot air oven at 120° to constant weight.

If great accuracy is desired, fold the filter carefully with the precipitate inside, wrap a platinum wire about it, hold it over a weighed platinum crucible, and burn the filter paper, allowing all the ash to fall into the crucible. Then ignite, weigh again, and take the weight of the ash from the weight of the dried substance.

URIC ACID DETERMINATION.

Principle: The uric acid is precipitated as ammonium urate which is then decomposed by the addition of H_2SO_4; and the liberated uric acid is titrated with N/20 potassium permanganate solution. To remove the mucoid substances always present, which would interfere with the subsequent operations, the urine is first treated with a reagent containing ammonium sulphate and uranium acetate. The resulting precipitate of uranium phosphate carries down the mucoid body. Ammonia is then added to alkalinize the solution and cause the precipitation of the ammonium urate.

Reagents: Solution of uranium acetate, made as follows:

Uranium acetate,	5 g.
Ammonium sulphate,	500 g.
Acetic acid, 10%,	60 c.c.
Water,	650 c.c.

Strong ammonia.
Ammonium sulphate solution, 10%.
N/20 potassium permanganate solution.
Strong sulphuric acid.

Technique: Into a tall beaker or cylinder measure 200 c.c. of urine and 50 c.c. of the uranium reagent. Allow to stand for one-half hour, and then decant, siphon, or filter off the supernatant fluid. Measure 125 c.c. (equal to 100 c.c. of urine) of this into a beaker, add 5 c.c. of strong ammonia, and set aside until the following day. Filter off the precipitate and wash with 10% ammonium sulphate solution until the filtrate is nearly or quite free from chlorides. Remove filter from funnel, open and wash the precipitate into a beaker with the ammonium sulphate solution. Add water to make 100 c.c. and dissolve the precipitate with 15 c.c. concentrated sulphuric acid. Titrate at once with the N/20 potassium permanganate solution. The end reaction is the first pink coloration extending through the entire liquid from the addition of two drops of permanganate solution while stirring with a glass rod, and remaining for 30 seconds.

Result: Each c.c. of permanganate solution used corresponds to 3.75 mg. of uric acid; add 3 mg. as correction due to solubility of ammonium urate.

1 mg. of uric acid = 0.000333 g. of N.

The amount usually present is 0.2 to 1.5 g. in 24 hours; that is, .5 to 2.5% of the total nitrogen.

CREATININ DETERMINATION.

Principle: On adding picric acid and sodium hydrate to a solution containing creatinin, a deep red color is produced. The intensity of this in the specimen of urine is compared with that of a standard solution of potassium bichromate. Sugar and albumin do not interfere; but acetone and diacetic acid, if present, must be removed by heating.

Reagents: N/2 potass. bichromate solution (24.55 g. per liter)
Sat. picric acid solution (about 12 g. per liter)
10% sodium hydrate solution.

Technique: Ten c.c. of urine is measured into a flask with a mark at 500 c.c., 15 c.c. of picric acid solution and 5 c.c. of sodium hydrate are added, and the mixture allowed to stand for five minutes. Pour a little bichromate solution into the two cylinders of the Duboscq colorimeter and set the left hand one to the 8 mm. mark. Then make several readings in order to accustom the eye to the colors. Now dilute the urine mixture to the 500 c.c. mark, and rinse out and half fill one of the cylinders with it, wipe off the glass rod, and then make several readings immediately and take the average. The reading must be made within ten minutes.

If the urine contains more than 15 mg. or less than 5 mg. of creatinin, repeat the determination with a smaller or larger amount of urine, as outside of these limits the determination is less accurate.

Calculation: $$10 \times \frac{8.1}{\text{Reading}} = \text{mg. creatinin.}$$

1 mg. creatinin = 0.000371 g. N.

The normal excretion of creatinin is about 20 to 30 mg. per kilo of body weight, fat persons yielding less and thin persons more. On an average diet the creatinin nitrogen equals about 3 to 5 per cent. of the total nitrogen.

UREA DETERMINATION.

Principle: Crystallized magnesium chloride boils in its water of crystallization at 160°C. At this temperature urea is quantitatively decomposed into ammonia and carbon dioxide in about one hour. If this is done in the presence of acid, the ammonia can subsequently be distilled off by the addition of an alkali, and the amount of urea calculated after the preformed ammonia, which must be separately determined, has been deducted.

Reagents: Concentrated HCl.
Crystallized magnesium chloride.
Paraffin.
1% aqueous solution alizarin red.
10% sodium hydrate.
Powdered pumice.
N/10 H_2SO_4.
N/10 NaOH.

Technique: Five c.c. of urine is measured with a finely calibrated pipette into an Erlenmeyer flask, and 5 c.c. concentrated HCl, 20 g. magnesium chloride, a piece of paraffin the size of a hazel nut, and 2 to 3 drops of alizarin red are added. The safety tube is inserted, and the fluid boiled briskly on a piece of wire gauze with asbestos center, until each drop falling from the safety tube produces a perceptible bump. The heat is then reduced and gentle boiling continued for a full hour. If the indicator in the flask turns red, several drops of the acid condensed in the bulb of the safety tube must be shaken back into the flask. At the end of an hour, the contents of the flask are transferred to a Jena glass distilling flask, washing the Erlenmeyer and condensing tube out carefully with hot water. About 700 c.c. of water is added to the distilling flask together with some powdered pumice and 20 c.c. of 10% sodium hydrate. The end of the condenser is fitted with a glass tube dipping below the surface of 50 c.c. N/10 H_2SO_4 and a few drops of Congo red in the receiver. The distillation is continued until the contents of the flask are nearly dry (at least an hour) and the distillate shows no ammonia with litmus paper. The distillate is then boiled for a few minutes to drive off the carbonic acid, is cooled, and titrated with N/10 NaOH, using Congo red as an indicator.

Result: The difference between the amount of N/10 acid in the receiver and the number of c.c. of alkali required for neutralization equals the number of c.c. of N/10 NH_3 yielded by the quantity of urine taken. From this result the preformed ammonia must be deducted, as well as the amount of ammonia in the magnesium chloride.

Each c.c. of N/10 $NH_3 = 0.0014$ g. of N, or 3 mg. of urea. The amount normally present on an average diet is about 20 to 40 g. and equals about 80 to 85% of the total nitrogen.

SULPHUR DETERMINATIONS.

Sulphur occurs in the urine in the following forms:

1 Inorganic or preformed sulphates:
2 Ethereal or conjugated sulphates in which H_2SO_4 is combined with aromatic compounds;
3 Neutral or unoxidized sulphur.

The partition of the three forms is determined as follows: The urine must be freed from albumin.

Reagents: Dilute HCl (1 part concentrated HCl to 4 parts H_2O by volume).
Barium chloride solution, 5%.
Benedict's solution:

Crystallized copper nitrate,	200 g.
Sodium or potassium chlorate,	50 g.
Distilled water to	1000 c.c.

INORGANIC SULPHATES.

Technique: Into an Erlenmeyer flask place about 100 c.c. of water; 10 c.c. of dilute hydrochloric acid; and 25 c.c. of urine. If the urine is dilute take 50 c.c. instead of 25, and a correspondingly smaller amount of water. Ten c.c. of barium chloride solution is added drop by drop from a pipette having a short piece of rubber tubing slipped over its upper end and provided with a screw pinchcock. The urine must not be disturbed while the barium chloride is being added. At the end of an hour or later, the mixture is shaken and filtered through a weighed Gooch crucible, as described below. The precipitate is washed with at least 200 c.c. of water. The crucible is then dried, ignited, placed in the desiccator, cooled, and then weighed.

The increase in weight indicates the amount of $BaSO_4$ referable to the inorganic sulphates in the amount of urine used.

1 g. $BaSO_4$ = 0.34301 g. SO_3.
1 " = 0.42015 g. H_2SO_4.
1 " = 0.13744 g. S.

To express the results in terms of SO_3, H_2SO_4, or S, multiply the weight of $BaSO_4$ by the proper factors as given above.

TOTAL SULPHATES

Principle: The ethereal sulphates are split by boiling with HCl, and the total sulphates resulting determined just as above.

Technique: Twenty-five c.c. of urine and 20 c.c. of dilute HCl (or 50 c.c. of urine and 4 c.c. of concentrated HCl) are gently boiled for 20 to 30 minutes in an Erlenmeyer flask, into which a funnel has been placed to reduce the loss of steam. The flask is cooled for about 2 to 3 minutes in running water, and the contents are diluted with cold water to about 150 c.c. Then precipitate with barium chloride, filter through a Gooch, and weigh as above.

ETHEREAL SULPHATES

The amount of these may be obtained by subtracting the amount of inorganic sulphates from that of the total sulphates.

The results obtained by these procedures will be in terms of barium sulphate. It is customary to report them in terms of SO_3; this is done by multiplying the amount of barium sulphate by the factor 0.34301. The total sulphur per 24 hours usually varies from 1 to 3.5 g. of SO_3. Of this the inorganic sulphates are about 85 to 90%; the ethereal sulphates, 8 to 10%; and the neutral sulphur about 5%; but the sulphur partition is largely modified by changes in diet.

TOTAL SULPHUR

Principle: All of the sulphur present is oxidized by heating with a reagent composed of copper nitrate and potassium chlorate. The former on heating decomposes into two vigorous oxidizing agents: nitrogen dioxide and cupric oxide, the latter forming a stable compound with the oxidized sulphur. This is dissolved in dilute hydrochloric acid, and the sulphur precipitated with barium sulphate and weighed.

Technique: Ten c.c. of urine is measured into a small (7 to 8 cm.) porcelain evaporating dish, and 5 c.c. of the reagent added. The contents of the dish are evaporated over a free flame, which is regulated to keep the solution just below the boiling point, so that there can be no loss through spattering. When dryness is reached the flame is raised slightly until the entire residue has blackened. The flame is then turned up in two stages to the full heat of the Bunsen burner and the contents of the dish thus *heated to redness for ten minutes after the black residue* (which first fuses) *has become dry.* This heating is to decompose the last traces of nitrate and chlorate. The flame is then removed and the dish allowed to cool more or less completely, 10 to 20 c.c. of dilute (1:4) HCl is then added to the residue in the dish, which is then warmed gently until the contents have completely dissolved and a perfectly clear, sparkling solution is obtained. This dissolving of the residue requires scarcely two minutes. With the aid of a stirring rod the solution is washed into a small Erlenmeyer flask, diluted with cold distilled water to 100 to 150 c.c., 10 or 20 c.c. of 10% barium chloride solution added drop by drop, and the solution allowed to stand for about an hour. It is then shaken up and filtered as usual through a weighed Gooch.

To make the Gooch crucible filter, pour a suspension of asbestos fiber in water into the crucible while strong suction is being applied, so that a firm felt work (about 2 mm. thick) is formed. The asbestos is prepared by scraping the crude material with a knife and adding the fibers to a large bulk of 5% HCl in a cylinder. Air is blown through to separate the fibers thoroughly, and the mixture is allowed to settle for a few minutes. The upper portion of the fluid containing the finer fibers is decanted and kept separately from the lower.

In making the filter the coarse material is poured on first and a little of the fine afterward. The filter is then washed by drawing distilled water through in a slow stream, is dried at 120°C., ignited, and weighed.

In igniting the barium sulphate precipitates the flame must not be applied directly to the bottom of the crucible, in order to avoid mechanical losses. The crucible is placed on a platinum crucible lid on a pipe stem triangle, and the outer, oxidizing flame of the Bunsen burner is used, first gently, and finally full force.

CHLORIDES.

Principle: The chlorides in a definite amount of urine are precipitated by a standard solution of silver nitrate in the presence of an excess of free nitric acid. The precipitate of silver chloride is filtered off, and the excess of silver remaining in solution is determined in the filtrate by titrating with a standard solution of potassium sulphocyanide, using a solution of iron alum as an indicator. As soon as the sulphocyanide has combined with all the silver to form white silver sulphocyanide, the deep red ferric sulphocyanide is formed and indicates the end reaction. In this way the amount of silver solution which combined with the chlorides is ascertained. Albumin need not be removed unless present in large amount.

Reagents required: $AgNO_3$ solution, of which 1 c.c. equals 0.01 NaCl.

Potassium sulphocyanide solution, of which 1 c.c. equals 1 c.c. of the $AgNO_3$ solution.

Saturated solution of ammonio ferric alum.

Nitric acid.

Technique: 10 c.c. of urine, accurately measured with a pipette, is placed in a flask with a mark at 100 c.c.; about 50 c.c. of water, 5 c.c. of nitric acid, and 20 c.c. of the silver solution are added. The nitric acid is to be measured in a graduate, but the silver solution must be accurately measured.

The mixture is well shaken, and distilled water is added to the 100 c.c. mark. The fluid is filtered through a small dry filter, and 50 c.c. taken, this amount corresponding to 5 c.c. of urine. This 50 c.c. is then poured into a beaker and the measuring flask rinsed into the same with distilled water.

Five c.c. of the alum solution is added, and the whole is titrated with the sulphocyanide solution to the appearance of the first reddish tinge.

Calculation: 20 minus the no. of c.c. of sulphocyanide solution used equals the no. of c.c. of silver solution required to precipitate the chlorides. Each c.c. of silver solution equals 0.01 of NaCl; therefore, 20 minus twice the reading, x 10 = milligrams of NaCl in 10 c.c. of urine. The usual amount in 24 hours is 10 to 15 grams.

DETERMINATION OF PHOSPHATES.

Principle: The urine is first treated with a solution of sodium acetate and glacial acetic acid in order to convert any monacid phosphate into diacid phosphate, and also to neutralize any nitric acid that may be formed during the subsequent titration, as this, if allowed to remain free, would cause partial solution of the precipitated uranyl phosphate. The titration is then performed with a standard solution of uranium nitrate which gives with phosphoric acid compounds in acetic acid solution a yellowish white precipitate of uranyl phosphate (UO_2HPO_4). As indicator cochineal is used, which gives a green color in the presence of an excess of uranium solution. Sugar and albumin do not interfere with the method. If the urine is deeply bile stained, it should be acidified with HCl and decolorized by the addition of a few crystals of potassium permanganate.

Reagents: 1 A solution of uranium nitrate of which 20 c.c. equals 0.1 g. P_2O_5.

2 Acetic acid solution:

Sodium acetate,	100 g.
Glacial acetic acid,	30 g.
Water to	100 c.c.

3 Tincture of cochineal.

Technique: Fifty c.c. of filtered urine is treated with 5 c.c. of the acetic acid mixture. A few drops of the cochineal indicator are added. The fluid is heated to boiling and titrated with the uranium solution until a trace of green color is seen, which does not disappear on stirring. The titration is repeated until accurately corresponding duplicates are obtained, and the result is calculated as follows:

$20 : 0.1 :: n : x$, n being the number of c.c. of uranium solution used.

The percentage of P_2O_5 equals 2x.

The usual amount of P_2O_5 on an average diet equals 1 to 5 g. in twenty-four hours.

AMMONIA DETERMINATION.

Principle: The ammonia is liberated by the addition of a weak alkali, is removed from the urine by the passage of a strong current of air, and is collected in N/10 sulphuric acid. The amount of the latter neutralized by the ammonia is then determined by titration with N/10 alkali.

Reagents: N/10 sulphuric acid.
N/10 sodium hydrate.
Sodium carbonate.
Congo red.
Powdered pumice.
Petroleum.

Technique: Place 25 c.c. of urine in a tall cylinder, and add about 10 c.c. of petroleum with a little powdered pumice to prevent foaming. In the receiving cylinder place 50 c.c. N/10 sulphuric acid and a few drops of Congo red. Insert the glass receiving tube and add sufficient water to cover its expanded portion. Add about 1 g. of sodium carbonate to the mixture in the urine cylinder and start the suction. Run pump at full strength 4 to 5 hours, and titrate with N/10 sodium hydrate to the first permanent red.

As the air of the laboratory usually contains ammonia it should be washed by causing it to pass through a cylinder containing dilute sulphuric acid before entering the urine.

Result: Divide the total amount of 24-hour urine by 25, and multiply the quotient by 50 minus the number of c.c. of N/10 NaOH required for neutralization.

1 c.c. N/10 $H_2SO_4 = 1$ c.c. N/10 NH_3 which contains 0.0017 g. NH_3 or 0.0014 g. N.

In normal urine the average amount is about 4% of total nitrogen, or about 0.7 g. in 24 hours.

SHAFFER'S METHOD FOR B-OXYBUTYRIC ACID.

From 25 to 250 c.c. of urine, depending upon the amount of b-oxybutyric acid expected, are measured into a 500 c.c. graduated cylinder.

Of diabetic and other urines giving a strong ferric chloride reaction for diacetic acid, when 5 to 10 g. or more of b-oxybutyric acid is expected, 25 to 50 c.c. or even less of urine will be a suitable quantity; when little or no acid is expected 125 to 250 c.c. may be used. In case the amount taken is sufficient for duplicate determinations such a quantity should be used as will yield 25 to 50 mg. of acetone from the b-oxybutyric acid.

An excess of basic lead acetate and from 10 to 20 c.c. of concentrated ammonium hydroxide are added. The mixture is diluted to the 500 c.c. mark, well shaken, and, after a few minutes, filtered.

A solution containing 10 per cent. of basic lead acetate is used, being shaken before use. A sufficient quantity must be added so that the filtrate gives a precipitate of lead sulphate upon acidification with sulphuric acid. When little or no sugar is present from 0.5 to 1 volume of the amount of urine used will generally be sufficient. When considerable sugar is present from three to four volumes may be necessary.

An aliquot part of the filtrate,* usually 250 c.c., is approximately neutralized with concentrated sulphuric acid and filtered; 200 c.c. of this filtrate is placed in an 800 c.c. Kjeldahl flask, 15 c.c. of concentrated sulphuric acid and a little talcum are added, and the mixture is distilled until 200 to 250 c.c. of the distillate are collected. (Distillate A.) The flask must be fitted with a dropping funnel and be marked with a blue pencil to show volumes of 250, 350, 500, and 600 c.c.

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* An aliquot part of the filtrate, usually 200 c.c. may be placed in an 800 c.c. Kjeldahl flask, neutralized with conc. sulphuric acid, an excess of 15 c.c. of conc. sulphuric acid added, the solution diluted with water to 600 c.c., and the mixture distilled for distillate A. If the excess of the basic lead acetate used has been large, a little difficulty with bumping and with determining when sufficient dichromate is present in the second distillation may be experienced.

Distillate A contains the acetone, preformed and from the diacetic acid, and also the volatile fatty acids. To remove the latter, including the disturbing formic acid, the distillate is washed into a 500 c.c. Kjeldahl flask, 10 c.c. of 10 per cent. sodium hydroxide are added, and the mixture is re-distilled. This distillate A, 250 to 300 c.c., is titrated with iodine and sodium thiosulphate.

The residue of urine, plus sulphuric acid, from which distillate A was obtained is diluted to 600 c.c. with water, 5 c.c. of a 5 per cent. solution of potassium dichromate are added, and the mixture is re-distilled until 500 c.c. of distillate are collected. Sufficient of the dichromate solution is added from time to time during the distillation to keep the solution from turning green. A few c.c. are added at a time, and care should be taken not to add it faster than is needed. Water is run in during the distillation so as to keep the volume of the distilling mixture at from 350 to 500 c.c. until near the end of the distillation, when it is allowed to decrease to about 200 c.c., but never lower than this volume.

One-half of this distillate B is placed in a 500 c.c. Kjeldahl flask, 10 c.c. of 10 per cent. sodium hydroxide are added, and 300 c.c. of the distillate (B) are collected. This is titrated with iodine and sodium thiosulphate as usual and gives the acetone from the b-oxybutyric acid.

After the collection of the distillate from the dichromate distillation, more water and dichromate may be added, and the distillation continued to see if all the b-oxybutyric acid has been oxidized.

103.4 per cent. N/10 solutions of iodine and sodium thiosulphate are used. One c.c. of the iodine solution = 1 mg. of acetone and 1.794 mg. of b-oxybutyric acid.

REPORT OF THE ST. LUKE'S HOSPITAL LABORATORY FOR THE YEAR 1910.

F. C. WOOD, M. D., DIRECTOR.

The following very much abbreviated report gives the statistical results of the work done in the laboratory in the various departments.

A number of the more important autopsies and surgical cases have been published during the year in various journals. Others will be published later.

DEPARTMENT OF BACTERIOLOGY.

The routine bacteriological examinations made during the year may be classified as follows:

Blood Cultures:

Typhoid bacillus	27	
Streptococcus	11	
Staphylococcus	7	
Negative	102	
Total		147

Urine Cultures:

Colon bacillus	20	
Typhoid bacillus	3	
Staphylococcus	8	
Streptococcus	1	
Mixed cultures	8	
Negative	35	
Total		75

Throat Cultures for diphtheria bacillus:

Positive	138	
Negative	300	
Total		438
Miscellaneous Cultures		100

Smears examined for Tubercle Bacillus:

Sputum:	Positive	107	
	Negative	352	459
Urine:	Positive	0	
	Negative	55	55
Chest and abdominal fluids:	Positive	4	
	Negative	15	19
Spinal fluid:	Positive	8	
	Negative	18	26
Stools:	Positive	0	
	Negative	15	15
			574
Guinea pigs inoculated for tubercle bacillus:	Positive	5	
	Negative	20	25

Vaginal smears examined for gonococcus	609
Urethral smears examined for gonococcus	18
Miscellaneous smears examined	81

The only serological examinations which have been made in any numbers are Wassermann reactions and Widals. A few of the Wassermann reactions were done with specimens sent from other clinics:

Wassermann reactions	305
Widal reactions	232

DEPARTMENT OF CLINICAL PATHOLOGY.

The following routine specimens were examined during the year:

Abdominal fluids	13
Blood:	
Estimation of coagulation time	5
" " hemoglobin	771
" " red cells	476
" " white cells	3,301
Examinations for malarial parasites	40
" " filaria	1
Chest fluids	63
Duodenal contents	2
Gastric contents	168
Miscellaneous smears	3
Spinal fluids, total and differential counts	9
Sputa	2
Stools	279
Urines	19,002

DEPARTMENT OF SURGICAL PATHOLOGY.

In the course of the year 656 specimens of tissue were examined histologically. The diagnoses may be classified as follows:

TUMORS.

Adenoma of the breast	2
Adenocarcinoma of the breast	1
Adenocarcinoma of the colon	3
Adenocarcinoma of the liver	1
Adenocarcinoma of the mesenteric nodes	2
Adenocarcinoma of the rectum	7
Adenocarcinoma of the stomach	2
Adenocarcinoma of the thyroid	1
Adenocarcinoma of the uterus	1
Carcinoma of the axillary nodes	4
Carcinoma of the back	2
Carcinoma of the bladder	1
Carcinoma of the breast	37
Carcinoma of the cervix uteri	5
Carcinoma of the corpus uteri	1
Carcinoma of the lymph nodes	4
Carcinoma of the neck, pigmented	1
Carcinoma of the ovary	3
Carcinoma of the peritoneum	2
Carcinoma of the skin of chest	1
Carcinoma of the splenic flexure	1
Carcinoma of the testicle	1
Carcinoma of the thigh	1
Carcinoma of the urethra	1
Carcinoma, gelatinous, of the axillary nodes	2
Carcinoma, gelatinous, of the intestine	1
Carcinoma, gelatinous, of the rectum	2
Carcinoma of the uterus	1
Chondroma of the scapula	1
Chordoma of the sacrum	1
Cystadenoma of the breast	4
Cystadenoma of the ovary	1
Epithelioma of the back	1
Epithelioma of the cervix uteri	9
Epithelioma of the cheek and face	9
Epithelioma of the eyelid	5
Epithelioma of the forehead	4
Epithelioma of the inferior maxillary gland	1
Epithelioma of the jaw	1
Epithelioma of the lip	9
Epithelioma of the orbit	1
Epithelioma of the penis	1

Epithelioma of the popliteal space	1
Epithelioma of the tongue	1
Epithelioma of the vagina	1
Epithelioma of the vulva	1
Epulis	2
Fibroadenoma of the breast	11
Fibroadenoma, intracanalicular, of the breast	5
Fibroangioma of the gum	1
Fibroma of the breast	2
Fibroma of the cheek	1
Fibroma of the gum	1
Fibroma of the jaw	1
Fibroma of the scalp	1
Fibromyoma of the uterus	74
Fibromyoma, with sarcomatous changes, of the uterus	1
Fibromyosarcoma of the broad ligament	1
Fibrosarcoma of the finger	1
Fibrosarcoma of the nose	2
Hypernephroma of the forearm	1
Hypernephroma of the kidney	1
Lipoma of the back	1
Lipoma of the buttock	1
Mixed tumor of the kidney	1
Mixed tumor of the parotid	1
Myxosarcoma of the liver	1
Papilloma of the larynx	1
Papilloma of the lip	1
Papilloma of the neck	1
Papilloma of the uvula	1
Papilloma of the vulva	1
Polyp of the cervix uteri	1
Polyp of the endometrium	1
Sarcoma of the abdomen	3
Sarcoma of the antrum	1
Sarcoma of the axilla	1
Sarcoma of the chest wall	1
Sarcoma of the groin	1
Sarcoma of the kidney	1
Sarcoma of the jaw	2
Sarcoma of the neck	4
Sarcoma of the ovary	1
Sarcoma of the tibia	1
Sarcoma of the tonsil	1
Sarcoma, melano, of the eye	1
Sarcoma, osteoid, of the ankle	1

CYSTS.

Branchial cleft cyst	1
Cyst of finger	1

Cyst of neck	1
Cyst of peritoneum	1
Cyst of thyroid	2
Dermoid cyst of ovary	1
Epidermoid cyst of supraorbital region	1
Intraligamentous cyst of ovary	2
Multilocular cyst of ovary	6
Papillary cyst of ovary	2
Parovarian cyst	3
Tubo-ovarian cyst	2
Unilocular cyst of ovary	5

MISCELLANEOUS TISSUES.

REPRODUCTIVE SYSTEM—FEMALE.

Cervix uteri, glandular erosion of	2
Cervix uteri, hyperplasia of	1
Decidua and chorionic villi,	17
Ectopic gestation	9
Endocervicitis, chronic	4
Endocervicitis, glandular	12
Endometritis, chronic	7
Endometritis, subacute	1
Endometrium, hyperplasia of	27
Hematosalpinx	1
Hydrosalpinx	6
Mastitis, chronic	2
Mastitis, tuberculous	1
Oophoritis, acute	2
Oophoritis, chronic	60
Ovary, edema of	3
Pyosalpinx	15
Salpingitis, acute	13
Salpingitis, chronic	25
Salpingitis, subacute	10
Salpingitis, tuberculous	5
Salpingo-oophoritis, acute	6
Salpingo-oophoritis, chronic	18
Salpingo-oophoritis, subacute	3
Vagina, tuberculosis of	1

REPRODUCTIVE SYSTEM—MALE.

Epididymis, tuberculosis of	1
Hydrocele sac	2
Prostate, adenomatous hyperplasia of	4
Prostate, hypertrophy of	3
Prostate, tuberculosis of	1
Scrotum, abscess of	2
Testicle, syphilis of	2
Testicle, tuberculosis of	4
Venereal wart	1

GASTROINTESTINAL SYSTEM.

Anus, ulcer of	1
Duodenum, inflammation of	1
Cholangitis, subacute	1
Cholecystitis, acute	2
Cholecystitis, chronic	4
Colitis, acute	1
Enteritis, chronic	1
Hepatitis, subacute interstitial	1
Peritonitis, acute	4
Peritonitis, tuberculous	3
Proctitis, chronic	1
Rectal mucosa, inflammation of	1
Tonsil, acute inflammation of	1
Tonsil, hypertrophy of	2

URINARY SYSTEM.

Kidney, parenchymatous degeneration of	1
Kidney, tuberculosis of	1
Pyelonephritis	2

BONES AND JOINTS.

Arthritis, villous	1
Bursitis, acute	1
Bursitis, chronic	1
Dactylitis, tuberculous	2
Exostosis of skull	1
Osteitis, chronic	2
Osteitis, productive, of thumb	1
Osteitis, rarefying	1
Osteomyelitis, tuberculous	1
Synovitis, acute	1
Synovitis, tuberculous	3
Tuberculosis of knee	1
Tuberculosis of metacarpal bone	1
Tuberculosis of radius, ulna, and carpal bones	1

LYMPH NODES.

Adenitis, simple	24
Adenitis, tuberculous	23
Hodgkin's disease	1

MISCELLANEOUS.

Blood clot	2
Connective tissue	4
Cutaneous tissue, chronic inflammation	1
Endarteritis	1
Endarteritis with gangrene of leg	4

Endophlebitis	2
Goiter, colloid	3
Goiter, exophthalmic, with glandular hyperplasia of thyroid	1
Granulation tissue, simple	10
Granulation tissue, tuberculous	4
Sinus, sacrococcygeal	3
Sinus, tuberculous, of face	1

POST-MORTEM EXAMINATIONS.

During the past year thirty-six autopsies were performed. The number during the summer months was somewhat smaller than usual as, owing to the reconstruction of the pathological laboratory, the regular autopsy room was not available. Some of the cases were of rather unusual interest.

804. Partial autopsy. Case of chronic cardiac disease, which had been under observation in the hospital for some time. The case was of interest because of a fairly large aneurism of the aorta which was entirely latent. The patient's symptoms were apparently due to the cardiac insufficiency and death was undoubtedly due to this cause.

Anatomical Diagnosis: Aneurism of the descending portion of the arch of the aorta with erosion of the vertebrae; arteriosclerosis; aortic insufficiency; chronic fibrous myocarditis; relative mitral insufficiency; slight chronic diffuse nephritis; chronic passive congestion of viscera.

805. Anatomical Diagnosis. Tuberculosis of right hip joint and spine; tuberculous psoas abscess, right and left, with perforation into descending colon; tuberculosis of bronchial lymph nodes and left lung; chronic adhesive pleurisy of left lung; fatty infiltration of liver; anemia of viscera. The case shows an interesting and unusual sequel of a psoas abscess—rupture into the colon.

806. Case of myocarditis of a common type, with less marked valvular lesions.

Anatomical Diagnosis: Chronic fibrous myocarditis; hypertrophy and dilatation of the heart; chronic mitral endocarditis; abscess and infarct of lung; hydrothorax; edema of brain; cirrhosis of liver; chronic diffuse nephritis; chronic passive congestion of viscera.

807. Case of a middle-aged woman who had suffered for a long time with symptoms of cardiac insufficiency, her illness running a chronic and uneventful course. Death was sudden and without warning. The autopsy showed the chronic cardiac and renal lesions usual in these cases with no anatomical cause for the sudden exitus except the rather striking dilatation of the right auricle.

Anatomical Diagnosis: Chronic diffuse nephritis; arteriosclerosis; cardiac hypertrophy and dilatation; relative tricuspid insufficiency; congestion and edema of lungs; cirrhosis of liver; chronic passive congestion of liver, spleen, and intestines.

808. Case of a child, two years old, who was admitted with subcutaneous emphysema of the neck and thorax which had appeared shortly before death. The autopsy showed that this was due to softening of a tuberculous bronchial lymph node which

had perforated into a bronchus. Air had been forced into the mediastinum by expiratory efforts and up through the suprasternal notch into the subcutaneous tissues, and also down along the aorta into the retroperitoneal tissues. There was also extensive miliary tuberculosis.

Anatomical Diagnosis: Perforation of bronchus by tuberculous lymph node; emphysema of mediastina, fascial planes of neck, chest, and abdomen, and of retroperitoneal areolar tissue; tuberculosis of mesenteric lymph nodes and miliary tuberculosis of lungs, spleen, kidneys, liver, and pancreas.

809. Case of chronic endocarditis with typical acute fibrinous pericarditis in a child of eleven. There was nothing unusual in the findings.

Anatomical Diagnosis: Chronic endocarditis with acute exacerbation; fibrinous adhesive pericarditis; pleurisy with effusion; fatty degeneration of liver; ascites.

810. Partial autopsy. Case of a congenital idiot, aged two and a half years. The brain, which was the only portion of the body examined, presented a remarkable picture. There was bilateral hydrocephalus, but the right hemisphere had collapsed, leaving a space between the cortex and the dura which was filled with arachnoid meshwork, the spaces in which contained a large amount of cerebrospinal fluid. The cortex of the left hemisphere was thinned out into a transparent membranous wall. There was very little cortical substance preserved in either hemisphere. In contrast to the degenerated condition of the forebrain, the medulla, corpora, pons, and cerebellum were well developed and apparently normal.

811. Case of acute lobar pneumonia in a man of sixty-six. The only feature of interest was the condition of the heart. There was a general arteriosclerosis, and the coronary involvement was extreme. The transverse branch of the left coronary was almost occluded near its origin by fibrous constrictions, and the marginal branch almost, if not entirely, obliterated. The left ventricular wall in the region supplied by this artery was much thinned out, and the muscle was almost completely replaced by fibrous tissue. In spite of this extreme muscular change, the heart was not markedly dilated, and compensation had apparently been fair.

Anatomical Diagnosis: Acute lobar pneumonia involving the left lower lobe; healed tuberculosis and atelectasis of left lung; chronic fibrous pleurisy with encysted empyema; atheroma of aorta, celiac axis, and coronaries; atrophy and fibrosis of myocardium; chronic endocarditis; chronic diffuse nephritis; chronic passive congestion of liver and spleen.

812. Case of acute lobar pneumonia in a woman of about thirty-five, whose meningeal symptoms had been very marked. At autopsy there was rather marked edema of the pia, but no exudate. The lesions otherwise were of the ordinary type.

Anatomical Diagnosis: Acute lobar pneumonia of the left lower lobe; chronic diffuse nephritis; congestion and edema of meninges; fibroid uterus.

813. This patient had, previous to admission, been operated on for hemorrhoids, and was brought into the hospital after sudden hemiplegia. At autopsy the longitudinal, lateral, and petrosal sinuses were filled with a red friable thrombus mass, and there was a small area of softening in the right parietal lobe. The hemorrhoidal veins were thrombosed. No plugs could be found in the cerebral arteries.

Anatomical Diagnosis: Thrombosis of cerebral sinuses; softening of brain; thrombosis of hemorrhoidal veins; right bronchopneumonia; cloudy swelling of liver and kidneys; fatty infiltration of liver.

814. Case of aneurism of the left gluteal artery in a man without marked arterial disease elsewhere. The aorta showed slight atheroma which was, however, no more extensive than is usually found in persons past middle age. The whole left gluteal artery was dilated, and beneath the gluteus medius formed an aneurism sac about 4 cm. in diameter. This had perforated some time before death and formed a large false aneurism which passed below the lower border of the medius muscle and extended from the upper level of the great trochanter to the ischial tuberosity beneath the panniculus,

Anatomical Diagnosis: Endarteritis; aneurism of gluteal artery with rupture and formation of false aneurism.

815. Case of strangulated femoral hernia without unusual features.

Anatomical Diagnosis: Gangrene of intestine; bronchopneumonia; chronic interstitial nephritis; arteriosclerosis; passive congestion of liver.

816. Case of purulent pericarditis and of partial obstruction of the intestine at the hepatic flexure due to fibrous band stretching from the great omentum to the right lobe of the liver.

Anatomical Diagnosis: Atrophy of left kidney, probably due to old pyonephrosis; perisplenitis; partial obstruction of gut due to band; cystitis; purulent pericarditis; congestion and edema of lungs; tuberculosis of right apex.

817. Case of tuberculous meningitis in a child of two years, with extensive tuberculosis of the mesenteric lymph nodes indicating the probable portal of entry. The bacilli in the spinal fluid were of the short plump form frequently assumed by the bovine type.

Anatomical Diagnosis: Tuberculous meningitis; tuberculosis of mesenteric lymph nodes.

817A. Case of thrombosis of meningeal artery in an old patient of fifty, without unusual features.

Anatomical Diagnosis: Edema of brain; small subdural hemorrhage, right side; small thrombus of middle meningeal artery; small area of necrosis in left temporal lobe; chronic diffuse nephritis, moderate; old tuberculosis of right lung; chronic cystitis.

818. Case of appendicitis and typhilitis, with perforation of the cecum and general peritonitis. The only unusual feature was the extensive involvement of the cecum.

819. Case of gastric carcinoma. The blood picture before death was of considerable interest on account of the large size of the red cells and some resemblance to the picture of pernicious anemia. The bone marrow from the femur was pink and on section showed many erythroblasts, some of which were distinctly of the megaloblastic type.

Anatomical Diagnosis: Carcinoma of the pylorus with metatasis in liver; pleurisy of right side with effusion; old tuberculosis of lung; extreme anemia; compensatory changes in bone marrow; chronic diffuse nephritis; chronic endocarditis.

820. Case of acute endocarditis and pericarditis. The heart showed advanced mitral stenosis and firm pericardial adhesions, the results of previous attacks.

Anatomical Diagnosis: Chronic endocarditis; mitral stenosis; acute septic endocarditis; serofibrinous pericarditis; infarct of lung; healed tuberculosis of lung and tuberculous pleurisy; chronic interstitial nephritis.

821. Case of hypertrophy of the pylorus in an infant of seven months. The muscle of the valve formed a firm ring 2 cm. in diameter and the lumen was constricted so as just to admit a probe. The mucosa of the canal lay in folds so that the constriction was evidently due to muscle spasm.

822. Partial autopsy. Case of acute suppurative meningitis involving both brain and cord, followihg an acute otitis media and mastoiditis. There was also a small thrombus in the torcular and another in the right lateral sinus.

823. Case of carcinomatosis secondary to carcinoma of the prostate, which had been removed five years previously.

Anatomical Diagnosis: Carcinoma of peritoneum, mesentery, and surface of intestine; metastases in retroperitoneal lymph nodes and fat; carcinoma of prostate (?); right inguinal colostomy; purulent peritonitis of right side of abdomen; acute right sided pleurisy; chronic diffuse nephritis of extreme grade; arteriosclerosis.

825. Case of typical status lymphaticus in an infant of eleven months. The child had succumbed to an attack of enteritis which so far as the anatomical lesions showed was not severe. The thymus measured 7.5 by 5 cm., and weighed 15 grams. The mesenteric lymph nodes were large, some of them measuring 0.5 cm. in diameter. The bronchial and superficial nodes were not enlarged. The Peyer's patches were somewhat hypertrophied, and the solitary follicles of the large bowel were much enlarged, forming nodules 2 to 3 mm. in diameter, which projected from the mucous surface of the gut.

827. Case of sarcoma of the brain involving the temporal lobe. The tumor was of the spindle cell type, the cells being arranged radially about the vessels in the manner seen in angiosarcoma. The growth was very vascular and showed considerable necrosis. The only other feature of interest was a huge retention cyst of the left kidney which entirely replaced the upper pole. The cyst was 7 cm. in diameter and was filled with clear fluid; its fibrous wall was 3 to 4 mm. thick. The kidneys were the seat of a chronic interstitial nephritis which was very moderate in extent.

Anatomical Diagnosis: Spindle cell sarcoma of brain; chronic interstitial nephritis; chronic myocarditis; healed tuberculosis of apices of both lungs with old pleural adhesions; edema and congestion of lungs; bronchopneumonia.

828. Case of purpura hemorrhagica. Anatomically there were found the usual hemorrhages in the skin and mucous membranes without hint as to their causation. There had been an extensive subserous hemorrhage involving two feet of the lower portion of ileum, which gave it the appearance of a thrombosed gut. No thrombus was found in the mesenteric vessels. The peritoneal fluid was slightly increased in amount, and was tinged with blood.

Anatomical Diagnosis: Purpura hemorrhagica; small abscesses in lung; extensive hemorrhages into last portion of ileum; small hemorrhages into mucous membrane of rectum and bladder.

829. Case presenting clinically the characteristic picture of carcinoma of the intestine with increasing emaciation and cachexia and alternating constipation and

diarrhea. At autopsy extensive ulcerations were found in the rectum and descending colon; the wall of the rectum was fibrous and thickened, but showed no evidence of new growth or of any specific type of inflammation. There were no impacted feces which could have caused the ulcers through pressure. Microscopically the ulceration involved the mucosa and partly the submucosa, the wall being formed by granulation tissue over the muscularis. The mucosa was undermined for some distance at the borders of the ulcers. There were great numbers of plasma cells throughout the granulation tissue, and some very large cells with vesicular nuclei, resembling degenerated ganglion cells.

Anatomical Diagnosis: Acute ulcerative proctitis and colitis; anemia and fatty degeneration of liver; congestion and interstitial inflammation of kidneys. Thorax was not examined.

830. Partial autopsy. Case of gliosarcoma of the brain involving the left occipital lobe. The tumor was very vascular and showed numerous areas of necrosis. The trunk was not examined.

832. Case of acute ulcerative endocarditis, in which a long-chain hemolyzing streptococcus was obtained by blood culture ante mortem. There were large cauliflower vegetations almost blocking the mitral orifice, and extensive villous vegetations on the septal wall of the left ventricle, and also on the left auricular wall. Both lungs were the seat of a typical central pneumonia; there were areas of consolidation in the stage of red hepatization involving the greater portion of both lungs; but the lung tissue immediately beneath the pleurae was not involved and formed a crepitant layer over the central consolidated mass.

Anatomical Diagnosis: Acute ulcerative endocarditis of mitral valve and auricular and ventricular wall; mural thrombi; cardiac hypertrophy and dilatation; mitral stenosis and insufficiency; relative tricuspid insufficiency; infarcts of lung and spleen; serous pericarditis and peritonitis; acute splenic tumor; cloudy swelling and congestion of liver; cloudy swelling of kidneys.

833. Case of a muscular man of middle age who died as the result of a fall down the shaft of an elevator. There were fractures of the skull involving the entire base, both orbits, and the petrous portion of both temporal bones. There was, however, no extensive hemorrhage into the skull. The outcome was independent of this lesion, and death was more probably due to rupture of the liver and right kidney. There was a huge ragged tear on the right lateral border of the liver extending deep into the liver substance. The fragmented liver tissue was exposed for an area 8 cm. in diameter. It appeared as if the organ had burst from sudden increase of pressure. This was of course followed by huge hemorrhage into the peritoneum. The right kidney was surrounded by a hematoma, and there was a tear in the kidney substance extending from the capsule to the pelvis. The 5th and 6th right ribs were fractured in the posterior axillary line. This fracture had no apparent connection with the ruptured viscera.

Anatomical Diagnosis: Contused wound of forehead; fracture of skull; fracture of ribs; rupture of liver and kidney with hemorrhage into peritoneum.

834. Case of huge cylindrical aneurism of the arch of the aorta, involving the transverse and descending portions. The sac was 10 cm. in diameter at the largest portion of the transverse arch. The wall was thick and fibrous with some calcareous plaques. Opening from the posterior wall of the transverse portion was a small thin-

walled saccular aneurism resting against the trachea just beneath the bifurcation. There was a small perforation into the trachea at this point, which was the immediate cause of death. It was remarkable that this patient had had no symptoms until a few weeks before, and then only cough with very slight dyspnea on exertion. It seemed probable that these symptoms were due to the development of the small saccular aneurism, which was in the region where aneurisms most promptly produce symptoms. The large cylindrical aneurism, which must have been of very long standing, apparently produced no symptoms whatever.

Anatomical Diagnosis: Cylindrical aneurism of aorta; perforation of trachea; congestion, edema, and emphysema of lungs with slight bronchopneumonia; general arteriosclerosis.

835. Case of hemopericardium due to rupture of coronary artery, or possibly to rupture of the ventricular wall. The heart showed an advanced fibrous myocarditis and extensive coronary sclerosis. The patient had been absolutely free from symptoms up to a few days before death, when she had a sudden attack of cardiac pain and dyspnea. These symptoms had entirely disappeared, when death occurred without warning. The coronary arteries were extensively diseased, the descending branch of the left coronary being practically occluded about 4 cm. from its origin by end- and mesarteritis. The left ventricular wall in the areas supplied by this artery was pale yellow in color and microscopically the muscle was found to be almost replaced by connective tissue. The hemorrhage occurred near the septum in the left ventricular wall near the apex, at the point where rupture of the heart is most frequent. On section of the heart, a thrombus was found in the apex of the ventricle. From this mass a thin strip of blood clot extended through a cleft in the muscle following the line of the muscle fibers downward to the left and finally backward, reaching the subpericardial fat posteriorly almost at the apex. On section, this thrombus was found to lie under the endocardium, and what at first appeared to be a rupture of the ventricle was probably a rupture of a coronary vessel which had perforated both internally beneath the endocardium and externally through the pericardium.

Anatomical Diagnosis: Coronary sclerosis and thrombosis; fibrosis of myocardium; rupture of left ventricle following rupture of coronary vessel; hemopericardium; arteriosclerosis; chronic diffuse nephritis; congestion, edema, emphysema, and healed tuberculosis of lungs; fatty degeneration of liver and kidney.

836. Examination of abdomen only. Case of gangrenous appendicitis with perforation and localized peritonitis.

837. Case of Hodgkin's disease, involving the tracheal and bronchial lymph nodes, spleen, liver, bone marrow, and retroperitoneal nodes; but without involvement of the cervical and superficial nodes. The case was of interest on account of the unusual distribution of the lesions and their anatomical character. There was much old hemorrhage into the enlarged nodes, so that many were deeply pigmented. The smaller nodules of the spleen were composed of round cells somewhat larger than lymphocytes with vesicular nuclei interspersed with eosinophiles and giant cells with one or more large vesicular nuclei. In the larger glands the lymphoid tissue was replaced in areas by fibrous connective tissue, very poor in cells. This fibrosis is not infrequently seen in Hodgkin's cases of long standing. About the hemorrhagic

areas there were great numbers of phagocytes filled with golden brown pigment. In the bone marrow the usual cellular constituents were almost completely replaced by the large round cells and giant cells of the Hodgkin's type.

Anatomical Diagnosis: Hodgkin's disease involving the tracheal, bronchial, and retroperitoneal lymph nodes, with nodules in liver and spleen and diffuse involvement of bone marrow; bilateral hydrothorax and hydropericardium; broncho-pneumonia.

838. Anatomical Diagnosis: Purulent peritonitis following myomectomy; parenchymatous changes in the organs; congestion and edema of lungs.

839. Case of chronic myocarditis and coronary sclerosis without unusual features, and with no anatomical cause for exitus, which was sudden. It showed rather strikingly the fibrous degeneration of the anterior wall of the left ventricle near the apex in the areas supplied by the descending branch of the left coronary artery. This branch was almost obliterated by arteritis.

Anatomical Diagnosis: Coronary sclerosis; chronic fibrous myocarditis; dilatation of the heart with relative mitral and tricuspid insufficiency; emphysema, congestion, and edema of lungs with right bronchopneumonia and healed tuberculosis; chronic interstitial hepatitis and pancreatitis; chronic adhesive pleurisy with effusion in the right pleura; chronic peritonitis; chronic diffuse nephritis with congestion; arteriosclerosis.

Out-patient Department.

METALLIC FOREIGN BODIES IN THE TISSUES; A NEW TELEPHONIC SEARCHER.

William S. Thomas, M.D.

Women at their housework sometimes lose a fragment of needle in hand or foot and seek relief in the surgical clinic or at the hands of their physician who may make a futile attempt to find and remove the offending object and finally send the patient to the hospital where the help of a skiagram can be had. In the search for a metallic foreign body the skiagram is an invaluable guide and when available no operation of the kind should be attempted without its aid unless the object sought for is unmistakably palpable through the skin. Even if so the incision is made cautiously and the parts handled gently in order to prevent pushing the foreign body from its position and losing it.

Operations for the removal of fragments of needles in the tissues are often provokingly difficult. In spite of a free incision and good retraction of the wound edges and a bloodless field, one may be reminded of the needle in the haystack. The eye is of less assistance than the finger in detecting the presence of a needle fragment. An infinitesimal amount of tissue between the foreign body and the surgeon's eye will conceal it whereas the sense of touch will reveal the object although covered by a layer of muscle or fascia.

If the fragment be small—say less than one half of an inch in length—and is causing no inconvenience, it may be left alone with impunity. Skiagrams not infrequently reveal the presence of a foreign body unsuspected by the patient. But as a rule muscular action, movement of the parts or contact of the skin with outside bodies causes pain or discomfort and makes proper the removal of the foreign body. Steel sewing needles are rather innocuous as sources of infection if left undisturbed. Perhaps their oxidization by contact with the body-fluids is accompanied by the production of germicidal substances. Needle fragments on removal are found to have lost their polish and to be blackened, even though but a short time has elapsed since their introduction.

If an operation be contemplated for the removal of a needle, the parts should be kept immobilized between the time of exposure to the Roentgen ray and that of operation. Otherwise it is not uncommon for the small sharp foreign body through muscular action, etc., materially to change its location.

Technic.—The usual preparation of the skin of the patient is followed by local anesthesia of the field and the application of an Esmarch's elastic bandage. With the skiagram as a guide, the incision is made as nearly as possible over the center of the needle fragment and at right angles to it, and should be long enough freely to admit the palpating finger, for even when the operation is quite bloodless the sense of touch will detect the presence of the foreign body before it can be seen. Good retraction should be maintained. The incision is carried cautiously through the fascia, and as nearly as will be allowed by the nature of the parts should be continued in the same plane in which the knife first enters. If the needle lies in the substance of a

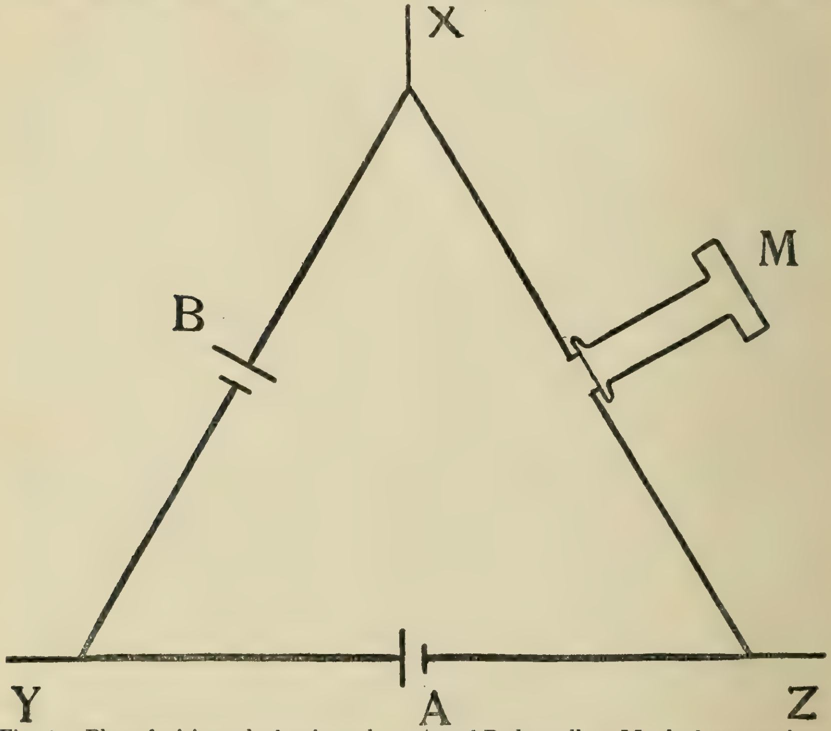

Fig. 1.—Plan of wiring telephonic probe. A and B, dry cells. M telephone receiver X Y Z, the three insulated needle.

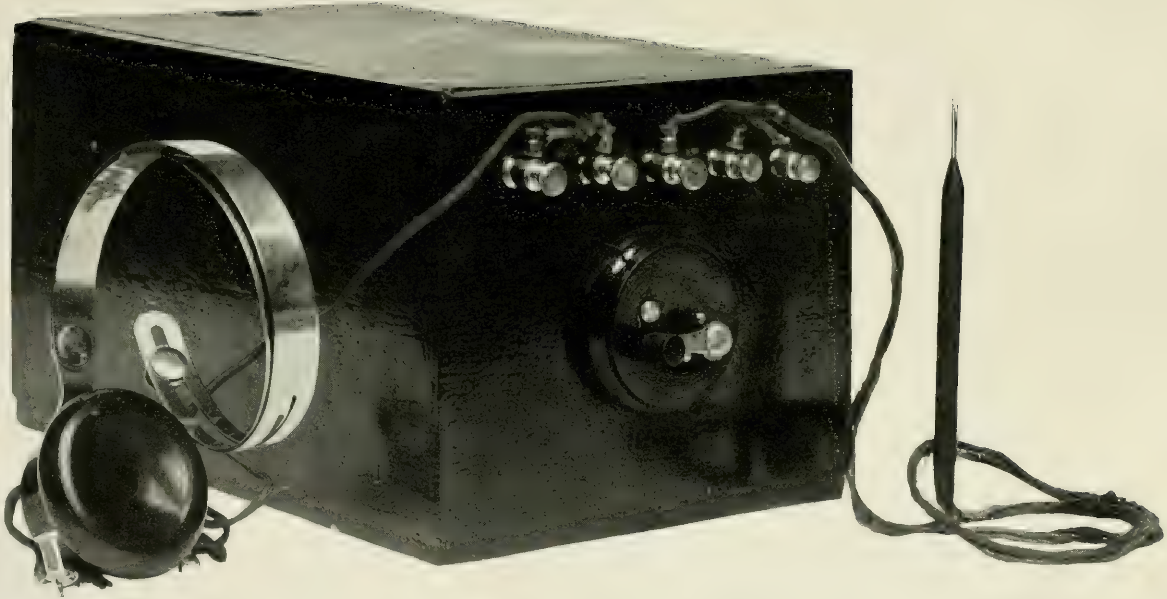

Fig. 2.—Telephonic searcher or probe at the right. Box containing dry cells connected with telephone receiver at the left.

muscle and parallel to its fibres it will be necessary of course to divide the muscle transversely. If however, the needle seems to lie transversely to the muscle fibres, these may be separated bluntly and may not need to be divided. The probe is of little use as a searcher because by its means it is impossible to differentiate a number of natural objects from a foreign body when the latter is separated from the probe by soft parts of any kind. The knife edge is an excellent detector but must of course, be used with caution as a searcher.

Once the needle is encountered it is immediately grasped with an artery forceps even though it is not completely exposed and though it may be necessary to grasp some soft parts with it. It is then pushed in the direction of its length until one end appears in the wound, when this in turn is grasped before the first hold is released and the fragment is removed. If attention to these details be not observed there is the likelihood of losing the fragment again or of breaking it during its removal. Cautiously cutting, frequently feeling with the finger tip, good retraction and a bloodless field are the essentials.

The occasional difficulties encountered in these seemingly trivial cases has led to the devising of an especial instrument to aid in finding fragments of needles or any metallic substances imbedded in the tissues. This consists of three parallel needles close together but nowhere touching each other and all secured in a handle. The needles are insulated from each other and each is connected with a wire of a three-conductor cord. These three conductors are connected with a battery of two dry cells and a telephone receiver in such a way that when once metallic contact is made between any two of the needles a sound is heard in the telephone receiver at the operator's ear. The illustration shows a box containing the dry cells and provided with switch and binding posts for connections. It also shows the telephone receiver with its head band and conducting cord and the searcher or probe with its needles in the hard rubber handle. The plan of the wiring is also shown in an illustration.

While the use of this instrument does not obviate the necessity of a skiagram it is of material assistance in finding a metallic foreign body and has made easy what might otherwise be a tedious task. Once the incision through the skin has been made the searcher is introduced if the anatomical relations permit of its use, and is carefully pushed to the length of the needles in the direction in which the foreign body is thought to lie. It is then withdrawn and is introduced parallel to and close by its first path until the neighborhood has been thoroughly searched. As will be seen, this instrument is used in much the same manner as the exploring needle, though with a finer adjustment, as it were.

Once the sound is heard in the telephone receiver informing the operator that a metallic body has been touched, the switch is turned off, to prevent heating at the point of contact by the electric current. The searcher in place is left there as a guide and the foreign body is easily cut down upon and removed.

The essential difference between this instrument and the older electric bullet probe lies in the fact that it is provided with more than two probing points and that these are sharp and will traverse soft parts independently of any sinus or wound of entrance.

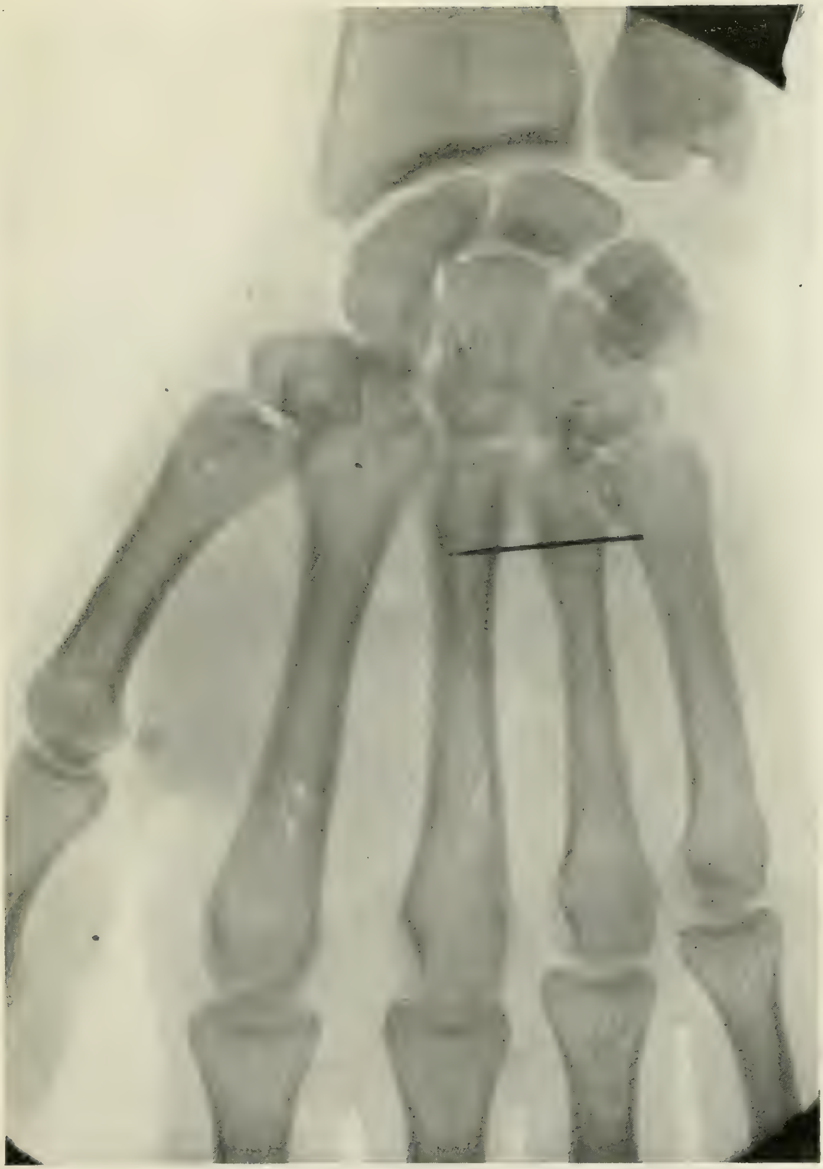

Fig. 3.—Fragment of needle shown by skiagram. Anterio-posterior aspect.

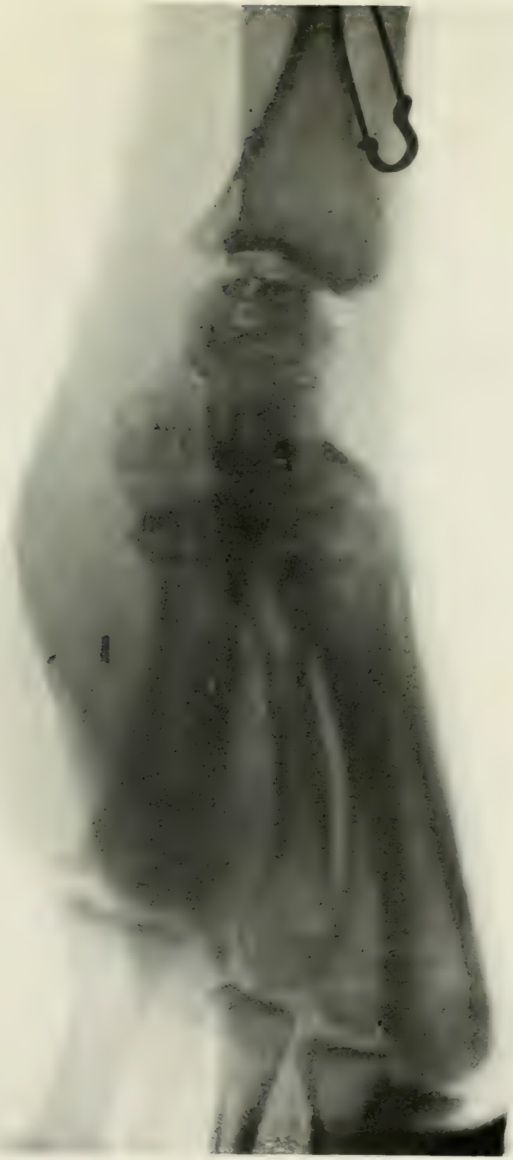

Fig. 4.—Fragment of needle shown by skiagram. Lateral aspect.

REPORTS OF CASES TREATED WITH TUBERCULIN.

From the Service of Austin W. Hollis, M.D.

M. Heminway Merriman, M.D.

The following report of cases is taken from those treated in the Tuberculosis Clinic of St. Luke's Hospital during the last three years. We endeavor to show the result of the tuberculin treatment, and incidentally the difficulties of this treatment in a large city clinic which is devoted to the care of those suffering from tuberculosis.

In all, 408 cases of tuberculosis have been treated in the clinic. Of these cases 94 or 23.4% have been considered suitable for the tuberculin treatment. Of these 94 cases, only 38 or 41% took more than ten injections of tuberculin. That is, out of a total of 408 cases treated in the clinic only 9.5% continued tuberculin treatment long enough for any conclusions whatever to be drawn.

It must be said that most of the favorable incipient cases were not put on the tuberculin treatment, because the great majority of these were sent to various sanitoria provided for such. A comparatively few early cases who, for various reasons, could not leave the city were put on the tuberculin treatment. All the cases chosen were afebrile or nearly so, and only included those patients who apparently could attend the clinic regularly.

All cases were begun with an initial dose of 1–10,000 mg. of bacillen emulsion, and this dose was increased by 1–10,000 mg. for ten doses, then by 1–1,000 mg, then by 1–100, etc. Injections were given twice a week, until 1–10 mg. was reached when they were given once a week.

All the later cases took their temperature every four hours and noted the degree of temperature in a book which was brought to the clinic for inspection, so that slight reactions could be noted. In 21 cases a well marked reaction took place at some period during the course of treatment. When this occurred, treatment was stopped until the temperature became normal, and then was resumed with smaller doses.

ON ADMISSION													AFTER TREATMENT					
Age	Duration of Symptoms	Cough	Haemoptysis	Fever	Sputum	Reaction	Weight	Temperature	Stage of Disease	Duration of Treatment	Number of Injections	Physical Signs	Case No.	Change in Symptoms	Change in Physical Signs	Weight	Final Result	Sputum
43		Severe	x	x		x	132	97–102	2	11 mos.	19	Infiltration of right upper and middle lobe		No symp.	Marked imp.	131	Cured	
22	2 mos.	Severe	0	x		x	168	97–102	2	6 mos.	31	Areas of consolidation and infiltration in both lungs		Prog.	Prog.	157	Admitted to hospital, unimp.	
39	10 mos.	Severe	0	x		x	142	98–100.4	3	3 mos.	10	Scattered infiltration in both lungs, breaking down of right upper		Prog.	Prog.	140	Died	
33	3 yrs.	Severe	x	x		0	111	97–99.2	2	5 mos.	21	Infiltration of one whole lung		Prog.	Prog.	118	Treatment stopped for non-attendance, unimp.	
20	10 wks.	Severe	x	x	Positive	0	127	97–99	1	11 mos.	36	Infiltration of one apex, dry pleurisy at base		No symp.	Imp.	140	Cured	
29	1 yr.	Severe	0	x		x	115	97–101	2	8 mos.	28	Infiltration of right upper and middle		Prog.	Prog.	119	Sent to hospital, unimp.	
30	3 yrs.	Severe	0	0		0	100	97–99	2	7 mos.	26	Infiltration of two lobes		Imp.	Imp.	130	Improved	
28	2 yrs.	Mod.	0	0		x	128	96–101	2	2 mos.	30	Infiltration of both apices		Prog.	Prog.	126	Left city unimp.	
43	1 yr.	Severe	x	x		x	101	96–104	2	11 mos.	39	Areas of consolidation in both lungs		Prog.	Prog.	93	Died	
20	10 mos.	Severe	0	0	Negative	x	131	97–100	2	8 mos.	20	Consolidation one lobe	8	Imp.	Imp.	130	Stopped because of work, imp.	Neg.
52	9 mos.	Mod.	x	x	Negative	0	141	97–100.2	2	9 mos.	60	Thickened pleura at base	11	No sym. for 3 mos.	No change	142	Apparently cured	Neg.
29	5 yrs.	Mod.	0	0	Negative	x	125	98–100	2	8 mos.	34	Infiltration of both upper lobes	13	No change	Imp.	119	Sent to Otisville	Neg.
42	8 wks.	Mod.	0	0	Negative	0	113½	98–99	2	9 mos.	60	Consolidation of one upper and infiltration of lower	16	Imp.	Marked imp.	118	Improved under treatment	Neg.
23	2 mos.	Severe	x	x	Positive	x	158	98–103	3	5 mos.	18	Marked consolidation of two lobes	18	Worse	Prog.	155	Admitted to hospital	Pos.
34	10 mos.	Mod.	x	x	Positive	x	168	97–103	3	4 mos.	20	Infiltration both lungs, with consolidation at one base	21	Worse	Prog.	167	Moved out of our district, unimproved	Pos.
39	2 mos.	Slight	x	0		0	124	98–100	2	6 mos.	17	Infiltration of one upper and one lower lobe	23	Marked imp.	Marked imp.	122	Left city improved	Neg.
39	2 yrs.	Marked	x	0	Negative	0	161	98–99	2	2 mos.	11	Slight infiltration of both upper lobes	29	Imp.	None	170	Improved, now working	Neg.
22	7 mos.	Mod.	0	0	Negative	0	185	97–99	1	5 mos.	20	Slight apical infiltration	35	Imp.	Imp.	186	Apparently cured, no signs found on discharge	Neg.
30	1 yr.	Slight	0	x	Negative	0	125½	98–99	2	3 mos.	11	Consolidation of two lobes with scattered infiltration on both lungs	39	Prog.	Prog.	117	Admitted to hospital	Neg.
35	3 mos.	Marked	0	0	Positive	0	165	97–100	2	4 mos.	15	Marked infiltration on both upper lobes	40	Imp.	No change	172	Arrested, now working	Pos.

ON ADMISSION													AFTER TREATMENT					
Age	Dur. of Symp.	Cough	Hemop.	Fever	Sput.	Reac.	Weight	Temp.	Stage	Dur. of Treat.	Inject.	Physical Signs	No.	Change in Symp.	Change in Phys. Signs	Weight	Final Result	Sput.
38	5 wks.	Mod.	0	0	Negative	0	123	97.3–100	1	6 mos.	10	Slight apical infiltration, and small pleural effusion at base	45	Imp.	Imp.	121	Arrested	Neg.
27	3 yrs.	Mod.	0	0	Positive	0	141	97.3–99	1	10 mos.	38	Marked apical infiltration	48	Imp.	Imp.	133	Imp., now working	Neg.
37	2 yrs.	Severe	0	0	Negative	0	112	97–99	2	7 mos.	31	Infiltration of one apex and one lower lobe	51	Imp.	Imp.	120	Imp., treatment stopped for surgical oper.	Pos.
42	6 mos.	Severe	0	x	Positive	x	151	97.3–102	2	8 mos.	12	Scattered infiltration both lungs	54	No. change	No change	139	Admitted to hospital	Neg.
53	1 yr.	Mod.	0	0	Negative	x	170	98–100	2	5 mos.	12	Infiltration of both apices, dry pleurisy one base	58	No. change	No change	174	Attendance irregular, treat. stopped, unimp.	Pos.
31	3 mos.	Mod.	0	0	Positive	x	145	98–100	1	3 mos.	10	Marked infiltration of one lobe	63	Imp.	Imp.	150	Imp., left city	
39	5 yrs.	Severe	x	0	Negative	x	155	98–101.2	2	14 mos.	75	Scattered infiltration, both lungs		No. change	Prog.	144	Still under treatment, unimp.	Neg.
26	1 mo.	Slight	0	0	Negative	x	105	97.2–100	1	6 mos.	22	Infiltration of one upper lobe		Imp.	Imp.	101	Stopped treatment, unimproved	Neg.
25	4 mos.	Severe	0	0	Positive	x	127	97–101	1	6 mos.	22	Slight apical infiltration		Same	More marked	135	Sent to Otisville	Pos.
40	1 yr.	Mod.	0	0	Positive	x	122	97.4–100	1	13 mos.	52	Marked infiltration of one upper lobe		Much imp.	Imp.	134	Still under treatment, doing well	Neg.
17	1 mo.	Severe	x	x	Positive	x	114	98-100	2	5 mos.	22	Several areas of infiltration in both lungs		Imp.	Marked imp.	118	Admitted to Otisville	Neg.
38	10 mos.	Mod.	x	x	Positive	x	144	98–101	2	3 mos.	16	Slight infiltration of one apex		Same	Same	149	Stopped because of surgical condition, unimp.	
39	6 mos.	Slight	x	x	Negative	x	123	97–100	2	16 mos.	74	Slight infiltration of both upper lobes		Marked imp.	Slight extension	122	Still under treatment	Neg.
27	6 mos.	Mod.	0	0	Positive	0	141	97–99	2	9 mos.	33	Marked infiltration of both upper lobes		Marked imp.	Imp.	140	Still under treatment, doing well	Pos.
42	5 mos.	Slight	x	x	Negative	0	155	98–99	2	3 mos.	17	Marked infiltration of one apex and severe asthma		Imp.	Same	172	Still under treatment, doing well	Neg.
40	4 mos.	Mod.	0	x	Negative	0	101	97–99	2	5 mos.	41	Infiltration of both upper lobes		Marked imp.	Slight imp.	112	Under treatment, doing well	Neg.
29	3 mos.	Slight	0	0	Negative	x	101	97–100	2	3 mos.	19	Infiltration of both upper and one lower lobe		Marked imp.	Less marked	107	Under treatment, doing well	Neg.
27	6 wks.	Severe	0	0	Positive	x	115	98–100	2	7 mos.	74	Infiltration of right upper and middle lobe		Marked imp.	Marked imp.	120	Under treatment, doing very well	Neg.

From the above cases it will be seen that the results were as follows: Cured, 2; Apparently cured, 2; Arrested, 2; Improved, 7; Unimproved, 8; Still under treatment, 9; Sent to hospital or sanitorium, 6; Died, 2.

In the foregoing tables a word of explanation may be needed.

"Duration of symptoms" refers to the time when the patient first noted symptoms which were probably due to tuberculosis.

"Fever" denotes that the patient had fever at times since the beginning of symptoms. In some of the early cases sputum examination were not made. From the time when the clinic became one of the Associated Tuberculosis Clinics all sputums had been examined monthly.

"Stage of disease" refers to the classification adopted by the National Association for the Study and Prevention of Tuberculosis.

I. Incipient—Slight initial lesion in the form of infiltration limited to a small part of one lobe. No tuberculous complications. Slight or no constitutional symptoms (particularly gastric of intestinal disturbances, or rapid loss of weight). Slight or no elevation of temperature. Expectoration small in amount or absent. Tubercle bacilli may be present or absent.

II. Moderately advanced.—No marked impairment of function, either local nor constitutional. Localized consolidation moderate in extent with little or no evidence of destruction of tissue, or disseminated fibroid deposits. No serious complications.

III. Far Advanced—Marked impairment of function, either local or constitutional. Localized consolidation intense, or disseminated areas of softening. Serious complications.

After treatment the results are classified as follows:

UNIMPROVED.	All essential symptoms and signs unabated or increased.
IMPROVED.	Constitutional symptoms lessened or entirely absent; physical signs improved or unchanged; cough and expectoration with bacilli usually present.
ARRESTED.	Absence of all constitutional symptoms; expectoration may or may not be present; physical signs stationary or retrogressive; the foregoing conditions to have existed for at least two months.
APPARENTLY CURED.	All constitutional symptoms, and expectoration with bacilli absent for a period of three months; the physical signs to be those of a healed lesion.
CURED.	All constitutional symptoms, and expectoration with the bacilli absent for a period of two years under ordinary conditions of life.

The cases whereof the following reports are made are numbered in the records of the Clinic as follows:—

For 1908-1909: 2, 9, 13, 14, 17, 30, 33, 38 and 66. For 1909-1910: 8, 11, 13, 16, 18, 21, 23, 29, 35, 39, 40, 45, 48, 51, 54, 58, 63, 66, 68, 69, 97, 106, 132, 141, 165, 170, 199, 229 and 343.

Conclusions—From the above report and from our experience, the therapeutic value of tuberculin may be assumed as positive and that it may be administered in out-door cases with safety, if supervision is careful, but reactions do occur even in small doses, although it may not be possible to assume that all reactions noted were due to tuberculin as under general treatment afebrile cases with well developed lesions frequently show temperature reaction.

There are many difficulties in carrying out this treatment in the out-door department of hospitals in large cities because of the impossibility of regular attendance and the bad housing conditions under which the cases live.

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